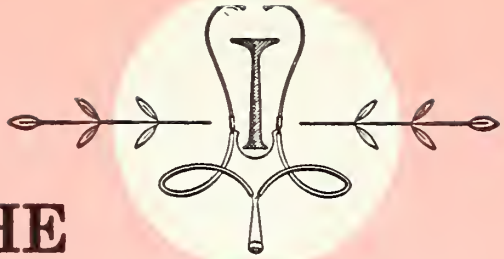

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THE Journal OF THE Kansas Medical Society

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The JOURNAL of the KANSAS MEDICAL SOCIETY

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CONTRAINDICATIONS. Administration of Antivert (meclizine HCl) during pregnancy or to women who may become pregnant is contraindicated in view of the teratogenic effect of the drug in rats.

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Meclizine HCl is contraindicated in individuals who have shown a previous hypersensitivity to it.

WARNINGS. Since drowsiness may, on occasion, occur with use of this drug, patients should be warned of this possibility and cautioned against driving a car or operating dangerous machinery.

Usage in Children: Clinical studies establishing safety and effectiveness in children have not been done; therefore, usage is not recommended in the pediatric age group.

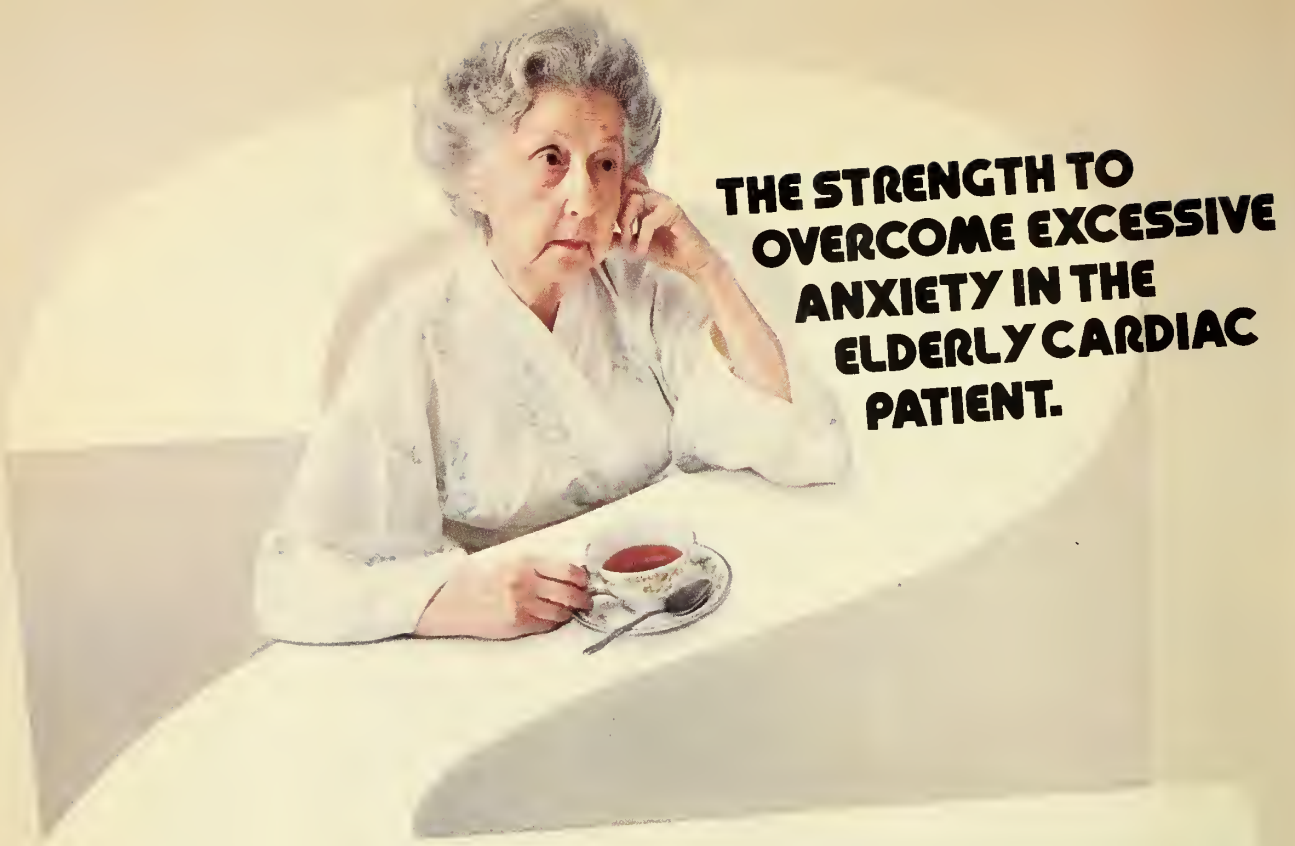
Usage in Pregnancy: See "Contraindications."

ADVERSE REACTIONS. Drowsiness, dry mouth and, on rare occasions, blurred vision have been reported.

More detailed professional information available on request.

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Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** When the combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome. Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium sparing action of triamterene is warranted. (See Box Warning.) Routine use of diuretics in healthy pregnant women is inappropriate; they are indicated in pregnancy only when edema is due to pathological causes.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

Supplied: Bottles of 100 and 1000 capsules; Single Unit Packages of 100 (intended for institutional use only).

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Auxiliary News

Traveling across Kansas during September, October, and November is an experience that I will always remember with a smile. The gracious hospitality and general friendliness of Kansans is sincere. West of Hutchinson, I renewed a habit of waving to every farmer and driver who did the same. My usual tendency to get lost didn't happen often and for that Jean Crouch and I were both thankful. I've often heard that the strength of Kansas is the people, which is true, but the beauty of Kansas this fall was the full glory of sunflowers and wild daisies spreading gold over the green grasses, and red sumac highlighting the hillsides.

Mrs. Crouch and I visited with members from 24 Auxiliary county chapters and in some instances had the pleasure of visiting with the physicians. We learned much. The programs, community projects, and service given by each Auxiliary chapter were varied and tailored to community needs. We were impressed by the originality and creativity of each. We look forward to visiting Reno, Central Kansas, Atchison, and Flint Hills chapters after the holidays.

In December, at the request of Chancellor Dykes, I attended a meeting wtih Drs. David Waxman, Joe Meek, Russell Mills and A. L. Murdock; Mr. Ralph Morgan, Director of Physician Recruitment; Mrs. John Doull, KUMC Hospital Auxiliary President; and Mrs. Pat Head, Executive Assistant, to review goals and to plan for continuing the Faculty Visits project. As you know, this project was begun while Dr. Kugel was Vice Chancellor. Seven faculty members, spouses and in some instances their children, have visited in six communities—Minneapolis, Herington, McPherson, Lawrence, Council Grove, and Shawnee Mission.

Reports from participating community families and KUMC and WSU faculty have been enthusiastically in favor of the person-to-person approach to developing better relationships between our medical school faculty and community physicians. It is an opportunity for the faculty to see firsthand the professional and living situations over the state. Hopefully, reverse visits will be possible in the future.

We plan to continue this program next spring. We want to facilitate for our faculty access into as many communities as possible. Instructors tend to influence their students; when they can relate favorable experiences to students about the people and towns of Kansas and practice potential in those towns, we think it will help to encourage young physicians and their families to settle and practice in our state.

Mrs. John Doull is contacting the faculty spouses in the Hospital Auxiliary and I look forward to working with her, Mrs. Head, and Vice Chancellor Waxman.

Doctor, we would like for you and your family to host a faculty family in your town and to show them what we have already told the committee is incomparable hospitality. A form is on page 31; please complete it and return it to me.

Sincerely,
Jackie Burnett
President
Kansas Medical Society Auxiliary

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*Librax has been evaluated as possibly effective for this indication.
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Please consult complete prescribing information, a summary of which follows:

Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants, causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

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Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

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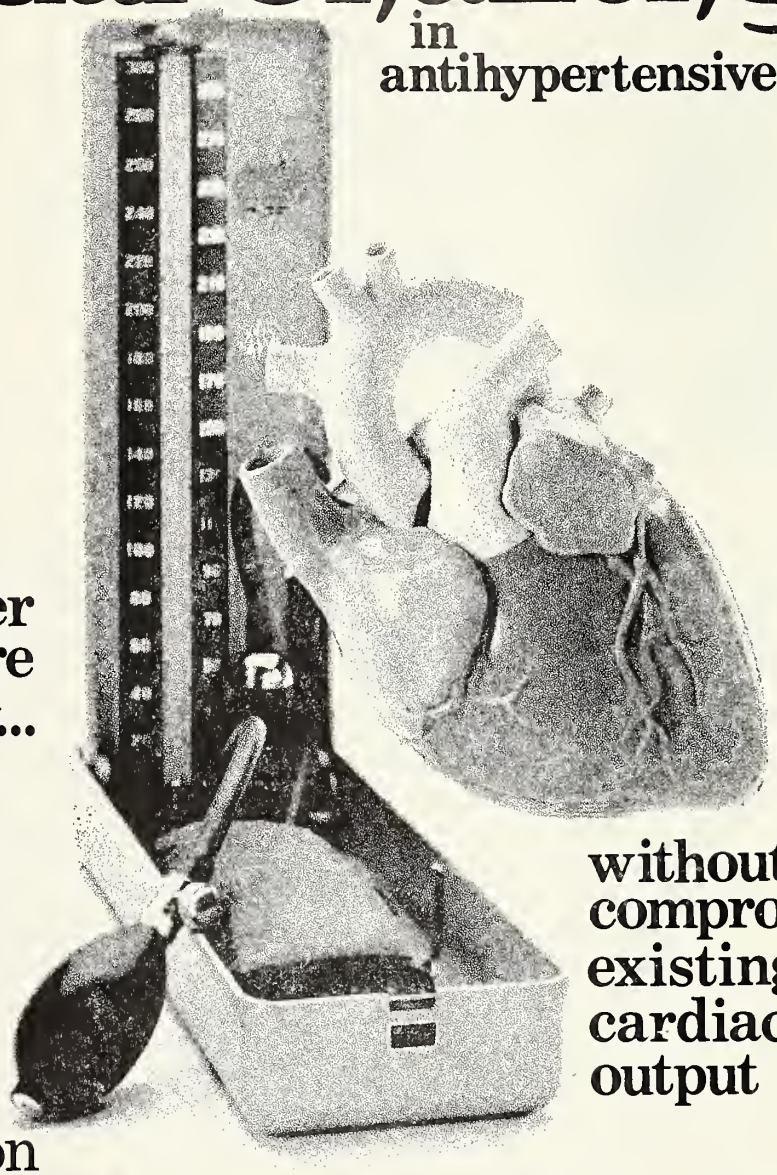
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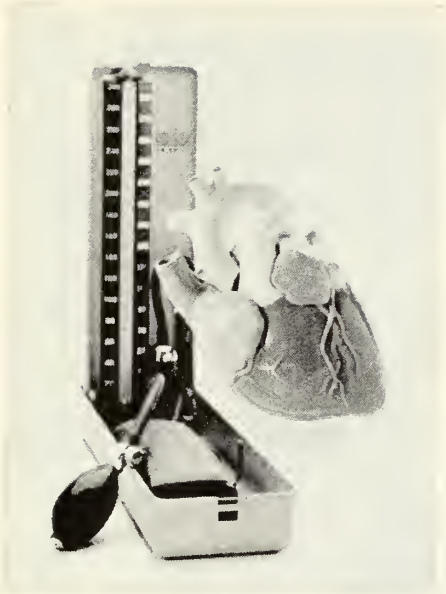
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For a brief summary of prescribing information, please see following page.

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ALDOMET®

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helps lower blood pressure effectively... usually with no direct effect on cardiac function– cardiac output is usually maintained

Contraindications: Active hepatic disease, such as acute hepatitis and active cirrhosis; if previous methyldopa therapy has been associated with liver disorders (see Warnings); hypersensitivity.

Warnings: It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. Read this section carefully to understand these reactions.

With prolonged methyldopa therapy, 10% to 20% of patients develop a positive direct Coombs test, usually between 6 and 12 months of therapy. Lowest incidence is at daily dosage of 1 g or less. This on rare occasions may be associated with hemolytic anemia, which could lead to potentially fatal complications. One cannot predict which patients with a positive direct Coombs test may develop hemolytic anemia. Prior existence or development of a positive direct Coombs test is not in itself a contraindication to use of methyldopa. If a positive Coombs test develops during methyldopa therapy, determine whether hemolytic anemia exists and whether the positive Coombs test may be a problem. For example, in addition to a positive direct Coombs test there is less often a positive indirect Coombs test which may interfere with cross matching of blood.

At the start of methyldopa therapy, it is desirable to do a blood count (hematocrit, hemoglobin, or red cell count) for a baseline or to establish whether there is anemia. Periodic blood counts should be done during therapy to detect hemolytic anemia. It may be useful to do a direct Coombs test before therapy and at 6 and 12 months after the start of therapy. If Coombs-positive hemolytic anemia occurs, the cause may be methyldopa and the drug should be discontinued. Usually the anemia remits promptly. If not, corticosteroids may be given and other causes of anemia should be considered. If the hemolytic anemia is related to methyldopa, the drug should not be reinstituted. When methyldopa causes Coombs positivity alone or with hemolytic anemia, the red cell is usually coated with gamma globulin of the IgG (gamma G) class only. The positive Coombs test may not revert to normal until weeks to months after methyldopa is stopped.

Should the need for transfusion arise in a patient receiving methyldopa, both a direct and an indirect Coombs test should be performed on his blood. In the absence of hemolytic anemia, usually only the direct Coombs test will be positive. A positive direct Coombs test alone will not interfere with typing or cross matching. If the indirect Coombs test is also positive, problems may arise in the major cross match and the assistance of a hematologist or transfusion expert will be needed.

Fever has occurred within first 3 weeks of therapy, occasionally with eosinophilia or abnormalities in liver function tests, such as serum alkaline phosphatase, serum transaminases (SGOT, SGPT), bilirubin, cephalin cholesterol flocculation, prothrombin time, and bromsulphalein retention. Jaundice, with or without fever, may occur, with onset usually in the first 2 to 3 months of therapy. In some patients the findings are consistent with those of cholestasis. Rarely fatal hepatic necrosis has been reported. These hepatic changes may represent hypersensitivity reactions; periodic determination of hepatic function should be done particularly during the first 6 to 12 weeks of therapy or whenever an unexplained fever occurs. If fever and abnormalities in liver function tests or jaundice appear, stop therapy with methyldopa. If caused by methyldopa, the temperature and abnormalities in liver function characteristically have reverted to normal when the drug was discontinued. Methyldopa should not be reinstituted in such patients.

Rarely, a reversible reduction of the white blood cell count with primary effect on granulocytes has been seen. Reversible thrombocytopenia has occurred rarely. When used with other antihypertensive drugs, potentiation of antihypertensive effect may occur. Patients should be followed carefully to detect side reactions or unusual manifestations of drug idiosyncrasy.

Pregnancy and Nursing: Use of any drug in women who are or may become pregnant or intend to nurse requires that anticipated benefits be weighed against possible risks; possibility of fetal injury or injury to a nursing infant cannot be excluded. Methyldopa crosses the placental barrier, appears in cord blood, and appears in breast milk.

Precautions: Should be used with caution in patients with history of previous liver disease or dysfunction (see Warnings). May interfere with measurement of: urinary uric acid by the phosphotungstate method, serum creatinine by the alkaline picrate method, and SGOT by colorimetric methods. Since methyldopa causes fluorescence in urine samples at the same wavelengths as catecholamines, falsely high levels of urinary catecholamines may be reported. This will interfere with the diagnosis of pheochromocytoma. It is important to recognize this phenomenon before a patient with a possible pheochromocytoma is subjected to surgery. Methyldopa is not recommended for patients with pheochromocytoma. Urine exposed to air after voiding may darken because of breakdown of methyldopa or its metabolites.

Stop drug if involuntary choreoathetotic movements occur in patients with severe bilateral cerebrovascular disease. Patients may require reduced doses of anesthetics; hypotension occurring during anesthesia usually can be controlled with vasopressors. Hypertension has recurred after dialysis in patients on methyldopa because the drug is removed by this procedure.

Adverse Reactions: *Central nervous system:* Sedation, headache, asthenia or weakness, usually early and transient; dizziness, lightheadedness, symptoms of cerebrovascular insufficiency, paresthesias, parkinsonism, Bell's palsy, decreased mental acuity, involuntary choreoathetotic movements; psychic disturbances, including nightmares and reversible mild psychoses or depression.

Cardiovascular: Bradycardia, aggravation of angina pectoris. Orthostatic hypotension (decrease daily dosage). Edema (and weight gain) usually relieved by use of a diuretic. (Discontinue methyldopa if edema progresses or signs of heart failure appear.)

Gastrointestinal: Nausea, vomiting, distention, constipation, flatus, diarrhea, mild dryness of mouth, sore or "black" tongue, pancreatitis, sialadenitis.

Hepatic: Abnormal liver function tests, jaundice, liver disorders.

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Allergic: Drug-related fever, lupus-like syndrome, myocarditis.

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Note: Initial adult dosage should be limited to 500 mg daily when given with antihypertensives other than thiazides. Tolerance may occur, usually between second and third months of therapy; increased dosage or adding a diuretic frequently restores effective control. Patients with impaired renal function may respond to smaller doses. Syncope in older patients may be related to increased sensitivity and advanced arteriosclerotic vascular disease; this may be avoided by lower doses.

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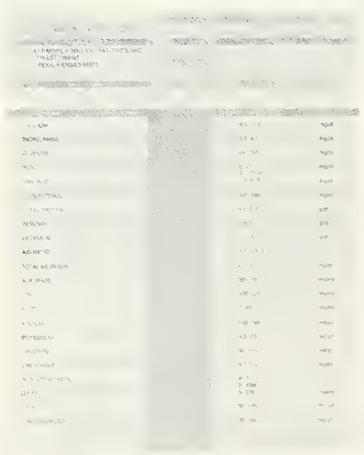
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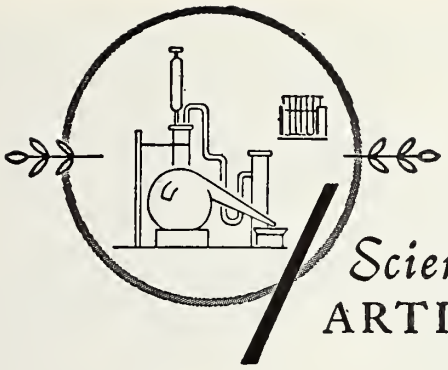
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Hydatidiform Mole

A Case With Coexistent Fetus

BYRON J. MASTERSON, M.D., ROBERT CORDER, M.D., DAVID A. LEITCH, M.D., R. CRAIG COLLINS, M.D. ***and*** **THOMAS E. SNYDER, M.D.,** ***Kansas City, Kansas***

HYDATIDIFORM MOLE is an uncommon developmental anomaly of the trophoblast occurring in the United States in approximately 1 : 2000 pregnancies.[1] The association of a fetus with a molar pregnancy is even more rare and few reports of this condition exist in the literature.[1] This case report is presented to add information to the established series of this unusual problem.

A case report of this uncommon developmental anomaly of the trophoblast is presented, and the diagnosis and treatment are discussed.

Case Report

A 27-year-old white female, gravida 3, para 3, abort 1, was referred to the University of Kansas Medical Center in the 16th week of pregnancy with a history of intermittent spotting for the previous two months. The bleeding became heavy three days prior to admission, requiring hospitalization. The patient had noted a recent increase in her abdominal girth. An x-ray picture of the abdomen obtained at her local hospital displayed a fetal shadow displaced over the right iliac crest. Because of her symptoms and the x-ray findings, an abdominal pregnancy was suspected. Physical examination on admission revealed a somewhat nervous female with an enlarged uterus compatible with approximately six months' gestation. Clinical findings revealed blood pressure of 140/90 mm Hg; pulse rate at rest, 104 beats/minute. Numerous telangiectases, which were blanched with pressure, were distributed over the chest. The thyroid was diffusely enlarged without nodules or bruit. Auscultation of the heart revealed a systolic ejection murmur which radiated to the carotid arteries. Pelvic examination disclosed a uterus consistent with a 27-week gestation, with severe anterior displacement of the cervix by a soft irregular mass in the cul-de-sac. Bilateral, tender, adnexal masses were present. A slight ooze of blood was noted at the external os.

The results of laboratory studies upon admission were normal except for: hemoglobin, 9.8 gm/100 ml; hematocrit, 28.3%; T4, 20.6 μg/100 ml (normal, 5-11 μg/100 ml); testosterone, 493 ng/100 ml (normal, 0-100 ng/100 ml). There was no protein present in the urine. A sonogram revealed an enlarged uterus containing a fetus (estimated age, 16 weeks) and another much larger mass thought to have characteristics of a molar pregnancy. The sonogram also confirmed the presence of two extrauterine cystic masses, one in the cul-de-sac and one in the adnexa on the right side. To confirm the diagnosis, a small amount of contrast material was injected directly into the amniotic sac under sonographic control. Following this procedure, a larger amount of contrast material was injected into uterine cavity, whereupon an abdominal film was tak-



Figure 1



Figure 2

en. The amniograms confirmed the diagnosis of an intrauterine pregnancy with a hydatidiform mole *(Figure 1)*.

An unsuccessful attempt was made on the following day to evacuate the uterus via vagina, and the mole and fetus were removed via exploratory coelotomy and hysterotomy *(Figure 2)*. Also noted at the time of surgery were ovaries containing large theca lutein cysts. About 2500 cc of molar tissue and blood were evacuated from the uterus. The patient did well postoperatively and was discharged from the hospital. Follow-up consisted of weekly HCG titers for ten weeks, and then monthly for one year. The titers quickly returned to zero, and there were no signs of persistence or recurrence of trophoblastic disease.

Discussion

The incidence of hydatidiform mole varies by racial and geographical factors. In general, the incidence of trophoblastic disease is most common in oriental populations and least common in Western nations. The incidence most often quoted in the United States is 1 : 2000 pregnancies for hydatidiform mole alone and from 1 : 10,000 to 1 : 100,000 for mole with coexistent fetus.[2]

Approximately 112 cases of mole with fetus have been reported in English literature. Beischer[1] in 1966 summarized the largest series of 92 cases. We add our case to 20 others reported since that time.

Our case illustrates several features seen when mole and fetus coexist. The uterine enlargement was greater than that corresponding to the period of gestation. Vaginal bleeding was severe enough to require transfusion, and some lower abdominal pain was experienced.[1] Pre-eclamptic symptoms were not prominent although the patient exhibited a diastolic pressure of 90 mm mercury at the time of admission. According to Beischer, second trimester pre-eclampsia was present in 21 of 92 cases.[1] A rather unusual feature was a suspected abdominal pregnancy. This situation has not been previously reported in the literature.

One of the more interesting features in our case is the presence of clinical hyperthyroidism with a pulse rate of 104 beats/minute and a systolic flow murmur. The true thyroxin level was 20.6 mg/100 ml with low to absent levels of TSH. The T4 rapidly returned to normal and the murmur disappeared. Hershman[3] has described a thyrotropin molecule which is immunologically different from TSH, chorionic thyrotropin of nor-

mal placenta, and long-acting thyroid stimulator (LATS). This molecule is thought to be produced by the molar tissue.

Our case also demonstrates the effect of high level of HCG on the ovaries. It is well known that the ovaries can manufacture testosterone. Samaan[4] studied the testosterone levels in women with trophoblastic disease, and his series of women with large ovaries at the time of surgery reflected testosterone levels over 700 ng/100 ml, with normal level at mid-trimester being 142 ng/100 ml. Our patient's testosterone level was 493 ng/100 ml prior to surgery and dropped rapidly postoperatively.

The characteristic pathological features of hydatidiform mole are:[5] (1) proliferation but not invasion of trophoblast; (2) hydropic degeneration of villi; and (3) inadequacy of blood vessels.

Initially, hydatidiform mole was thought to be solely a result of degeneration of a blighted ovum. Mole coexisting with a viable fetus was believed to be a result of the degeneration of one ovum in a biovular pregnancy; however, this does not explain some of the reported cases in which apparent diffuse degeneration of a single placenta occurred. Beischer[1] has reported five placental variations of mole with fetus:

1. Complete vesicular change of one of biovular twin placenta;
2. Extensive molar change in a single but possible uniovular twin placenta;
3. Complete hydatidiform degeneration of a single placenta;
4. Vesicular degeneration of a single cotyledon;
5. Diffuse hydatidiform change throughout a single placenta.

Our case probably falls into the first group, since there was an identifiable, normal-appearing placenta associated with the fetus.

This case also illustrates another pathological feature common to hydatidiform moles with the presence of large bilateral theca lutein cysts. The ovaries decreased to normal size within six weeks postoperatively.

The initial diagnosis was extrauterine pregnancy; however, molar disease was suspected. The diagnosis was simplified greatly by the use of gray mode sonography. In this technique, the echo amplitudes are displayed as different shades of gray. Gray scale sonography is much more sensitive than the B-mode and can illustrate soft tissue in much greater detail.[6]

Amniography was also used to confirm the diagnosis. There appears to be a low incidence of ill effects from this procedure; however, there is not enough information currently available to verify this.[7] Excellent results have been reported with sonography alone; however, amniography provides the definitive diagnostic study to confirm the presence of a molar pregnancy.[6]

Our patient was followed in the standard manner for all molar pregnancies at the University of Kansas Medical Center. Her regimen consisted of weekly HCG titers until zero titer was obtained for three consecutive weeks. (If titers do not decrease to zero within 60 days of evacuation, or if they rise after initially decreasing, or if they decrease somewhat and then stabilize, the patient is returned to the hospital for evaluation and possible intensive chemotherapy.) This patient's titers fell rapidly to zero and did not rise. It is very important that the patient not become pregnant during the follow-up period, since the elevated HCG of pregnancy would confuse the diagnosis.

Summary

A case of hydatidiform mole with coexistent fetus has been presented. This case demonstrates many of the symptoms of classical hydatidiform mole, as well as the assistance of gray scale sonography in the diagnosis of difficult obstetrical problems.

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Prinzmetal Angina

Long Term Follow-up After Recurrent Ventricular Arrhythmias

DOUGLAS B. BOGART, M.D.; H. DANIEL LEWIS, JR., M.D.; DANIEL R. HINTHORN, M.D. *and* RAYMOND A. SCHWEGLER, M.D.,

Kansas City*

THE PROGNOSIS in patients with Prinzmetal angina is grave. Silverman and Flamm in their review of the literature reported that more than one half of these patients sustained a myocardial infarction or died within one year.[1] There is little information in the literature on the long term prognosis for such patients. This report details the case of a patient with Prinzmetal angina who experienced 159 episodes of ventricular tachycardia and 52 episodes of ventricular fibrillation during a seven month period. The patient survived these episodes, and is now living—active and asymptomatic—five years following this illness.

The case of a patient who experienced multiple episodes of ventricular tachycardia and ventricular fibrillation during a seven-month period, and who is now living —active and asymptomatic—five years later, is presented and discussed.

Case Report

A male, age 46 years, was in his usual state of health until November 18, 1971, when he experienced a syncopal episode at work. He was admitted to a hospital where he was found to have unstable angina pectoris and recurrent ventricular tachycardia and fibrillation. He had 22 episodes of ventricular tachycardia and 14 episodes of ventricular fibrillation which required DC countershock. No myocardial infarction was documented at that time. Because of the continuing ventricular irritability, he was transferred to the Kansas City Veterans Administration Hospital on December 14. At the time of transfer, he was receiving propranolol 30 mg/6 hr, Nitro-Bid two times a day, and nitroglycerin as needed for angina.

Past history revealed a two year history of hypertension and a six month history of effort-related angina pectoris. There was no previous history of a myocardial infarction.

Physical examination revealed a well developed, well nourished white male in no distress with blood pressure 180/90 mm Hg, heart rate 60/minute and respiratory rate 12/minute. There was no jugular venous distension and the chest was clear. Cardiac examination revealed a fourth heart sound. Examination of the abdomen, extremities, and neurological system produced normal results. The admission electrocardiogram is shown in *Figure 1*. The chest roentgenogram taken on admission produced normal results.

The patient was admitted to the coronary care unit and continued to experience ventricular tachycardia and fibrillation. A repeating pattern began to emerge in that the MCL_1 lead would show ST segment elevation, the patient would develop typical angina pectoris, and then the ventricular arrhythmias would frequently occur. A typical sequence is represented by *Figure 2* which shows the monitor before, during, and after chest pain. These changes were confined to the anterior pericordial leads. It was observed that if nitroglycerin was given early in this sequence, the arrhythmias could occasionally be prevented. In addition, when the patient began to experience light-headedness from the ventricular tachycardia, he would frequently "thumpvert" himself by a sharp blow to the precordium.

Despite an extensive medical program which included nitroglycerin, long acting nitrites, propranolol, digoxin, procainamide, quinidine sulfate, lidocaine and phynytoin, the patient continued to have repeated episodes. A temporary pacemaker was placed on December 21. The episodes seemed to decrease slightly at a pacing rate of 90/minute. A permanent transvenous rate adjustable pacemaker was implanted on January 4. The patient continued to have ventricular arrhythmias despite variations in the pacemaker rate. The medications were again adjusted and included long acting nitrites, nitroglycerin, quinidine sulfate, propranolol,

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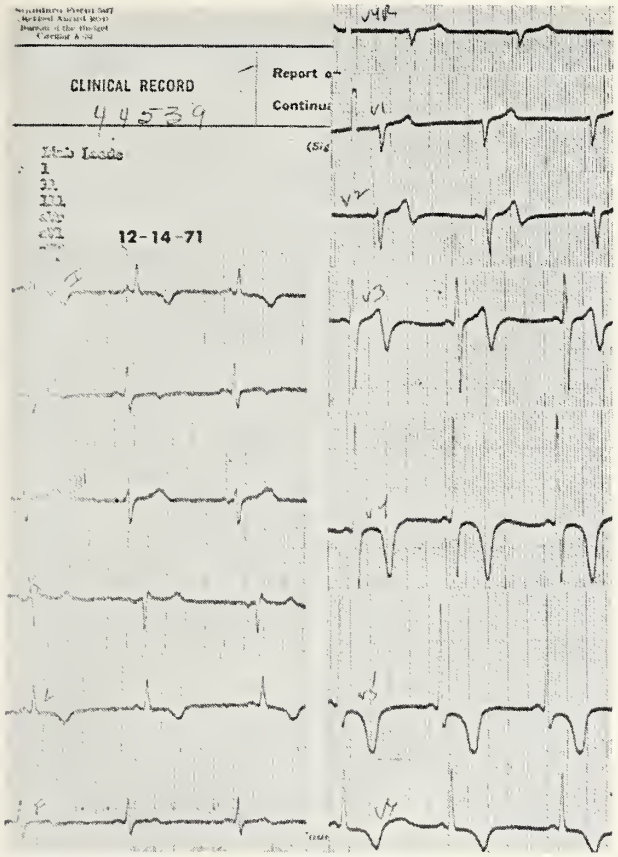


Figure 1. The admission electrocardiogram shows ischemic changes in the precordial leads with marked T wave inversion. There is no evidence for a transmural myocardial infarction.

chlorothiazide, digoxin, and potassium chloride. Despite pushing the medications to toxicity, the patient continued to have severe ventricular irritability. Because of this persistent problem, the patient had a cardiac catheterization on January 28. The left ventricular end diastolic pressure was 10 mm Hg. The left ventricular angiogram disclosed a small dyskinetic segment at the apex with an overall ejection fraction of 55 per cent. The left coronary artery was a small diffusely diseased vessel, but with no high grade localized obstructions. The right coronary artery had a 60 per cent midcourse obstruction with a good distal vessel. It was felt that these abnormalities were not suitable for a surgical approach.

With continued medical therapy, the episodes gradually began to decrease in frequency over the next six weeks. The last episode of ventricular fibrillation occurred on March 8. The patient did have 20 episodes of ventricular tachycardia following this, the final one occurring on April 28. At this time, the pacemaker rate was 85, and the medical program included digoxin

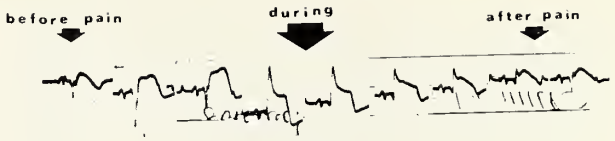


Figure 2. The MCL_1 lead showing the evolution of the electrocardiographic changes occurring before, during and after chest pain. Striking ST segment elevation is seen during chest pain.

0.125 mg/day, propranolol 10 mg/6 hr, furosemide 80 mg/2 days, and nitroglycerin as needed. The patient had experienced 159 episodes of ventricular tachycardia and at least 52 episodes of ventricular fibrillation which required DC countershock between November 18, 1971, and April 28, 1972. Throughout this time period there was no electrocardiographic or enzymatic evidence for an acute myocardial infarction.

The patient was continuously monitored in the coronary care unit until May 5 when he was transferred to the ward. He continued to have severe incapacitating exertional angina but no further Prinzmetal angina. The exertional angina severely impaired his ambulation. During May and most of June, he had from five to ten episodes of angina daily provoked by very minimal activity. The chest pain was improving at the time of his hospital discharge on July 3.

Following dismissal, the angina continued to decrease in frequency until it abated in October, 1972. He was followed as an outpatient until June, 1977; he is currently active with no angina pectoris or congestive heart failure. The pacemaker rate is 84/minute, and the medical program includes digoxin 0.125 mg/day, propranolol 10 mg/6 hr, hydrochlorothiazide 50 mg two times a day, alphamethyldopa 500 mg/6 hr, and potassium chloride.

Discussion

The criteria for diagnosis of Prinzmetal angina are recurrent angina at rest associated with ST segment elevation, return of the ST segments to control levels after the cessation of pain and no evolution of a myocardial infarction.[2] However, MacAlpin[3] has questioned these criteria in patients who present with a clinical history of unstable angina, such as the patient reported here. He feels that these patients are best considered to have unstable angina and not Prinzmetal angina. However, the exact classification of such patients is not fully defined.

The patient presented here would meet the criteria outlined by Prinzmetal *et al.*[2] His angina did progress from typical exertional angina, to angina at rest with ST segment elevation, to exertional angina which final-

ly subsided over an eleven month period of time. During the five month period when the angina was occurring with ST segment elevation the patient had refractory ventricular arrhythmia which did not respond to a wide variety of therapeutic maneuvers. This patient has been followed for five years and has had no recurrence of angina pectoris or ventricular arrhythmias.

Nitroglycerin has been reported to be effective prophylactically and therapeutically for Prinzmetal angina.[2] While this drug was of some help it did not adequately control the problem. All of the currently available antiarrhythmic agents were used in this patient, and despite pushing these drugs to toxicity the arrhythmias were not controlled.

Overdrive ventricular pacing was attempted in this patient, but despite wide variation in the pacing rate (70 to 120 beats per minute), he continued to experience significant ventricular arrhythmias. Johnson *et al.* have reported that this form of therapy is most effective when the prepacing heart rate is slow, which was not the case with this patient.[4]

Cardiac catheterization was performed in an attempt to find a surgically correctable lesion which could account for the patient's refractory symptoms. The lesions found at cardiac catheterization included a very small left ventricular aneurysm, a small diffusely diseased left coronary artery, and a 60 per cent obstructive lesion in the midcourse of the right coronary artery. Surgical therapy was therefore not undertaken in this patient as the electrocardiographic changes occurred in areas supplied by the left coronary artery. Coronary artery bypass has been reported to have limited value in patients with Prinzmetal angina who do not have proximal high grade obstructions.[5] Also, Oliva *et al.* have shown that the site of spasm may vary in an individual patient.[6] This finding may account for some of the poor surgical results which have been reported.

At the present time there is no specific therapy for Prinzmetal angina. Recently, Levene and Freeman[7] have reported the effectiveness of alpha-adrenergic blockade in a patient with coronary artery spasm. However, the efficacy of such an approach in patients with Prinzmetal angina has not been determined. Because there is no specific treatment for this entity, careful supportive therapy for these patients is indicated. With such an approach, our patient survived his catastrophic illness. The gradual improvement this patient experienced over a seven month period may suggest the development of collateral circulation. This concept was originally suggested by Prinzmetal *et al.* to explain the gradual loss of symptoms seen in some of their patients.[2]

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Hemiplegia

A Case of Unusual Etiology

VEMIREDDI N. KUMAR, M.D., ***Kansas City, Kansas***

A WHITE FEMALE, 83 years old, was admitted to the University of Kansas Medical Center with weakness of the left leg of one day duration. Before the onset of weakness, the patient was in good health. Past medical history included hysterectomy for carcinoma of the uterus 30 years prior to admission, and hemicolectomy for carcinoma of descending colon five months earlier.

At the time of admission, the patient was alert and well oriented. Cranial nerves were intact. There was weakness (50%) of the left upper and lower extremities and the deep tendon reflexes were slightly increased. Babinski's reflex was positive on the left side. Coordination of the left hand was impaired. Romberg was positive, and the patient tended to fall to the left. All the other sensations were intact. An initial diagnosis of left sided hemiplegia was entertained, possibly secondary to cerebrovascular accident (CVA) or metastatic lesion in the brain.

Routine laboratory testing produced normal results. Chest x-ray, bone scan, brain scan and lumbar puncture were normal. X-ray revealed degenerative changes involving the upper cervical spine.

On the fifth day, the patient lost control of the bladder and bowel, and weakness on the left side increased. Re-examination on the tenth day revealed decrease of pain and temperature sensations in the right leg. The remainder of the neurologic examination produced the same results as before. In view of the new clinical findings, the diagnosis was revised to spinal cord compression with Brown-Sequard syndrome. A cervical myelogram revealed no mass effect. There was mild widening of the spinal cord at C-4-5 which was considered a nonspecific finding. A neurosurgeon was consulted who suggested using Decadron and a towel splint for the neck. The patient's condition improved somewhat during the next ten days and then started deteriorating. Decompression cervical laminectomy was suggested, but the patient refused operation. In the next two days the neurologic impairment progressed to complete quadriplegia. On the 30th day it was noted that her intercostal muscles were paralyzed and the sensory level was at C-4. The next day the patient had diaphragmatic paralysis and died.

Autopsy Report

The spinal cord was cylindrically enlarged from nerve roots C-1 to C-6. A cross section of the cord at C-4 revealed metastatic neoplasm involving 50 per cent of the area of the spinal cord. Inspection of the spinal canal showed no evidence of bony intrusion on the contour of the canal. There was no neoplasm present in the subdural or epidural space. The microscopic picture of the metastatic neoplasm was that of a well differentiated adenocarcinoma consistent with primary carcinoma of the colon *(Figures 1-2)*. Accessory findings at autopsy include a minimal atherosclerosis of the abdominal aorta and mild pulmonary emphysema.

An unusual metastatic lesion of the cervical cord with signs and symptoms of hemiplegia, which resulted in death, is presented. The etio-pathologic process and the final outcome are discussed.

Discussion

The rarity of intramedullary spinal cord metastases is well documented by a review of the literature. Benson[1] in 1960 reported 27 cases including his own three cases. The infrequency of intramedullary cord involvement contrasts sharply with the frequency of

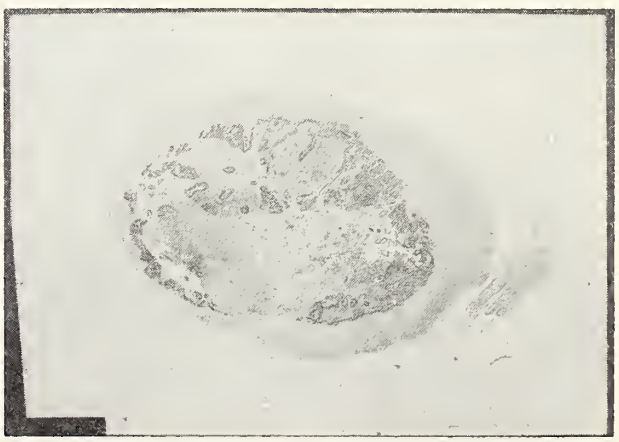


Figure 1. (H&E Stain × 5) Cross section of cervical spinal cord showing the metastatic neoplasm occupying the central half of the cord.

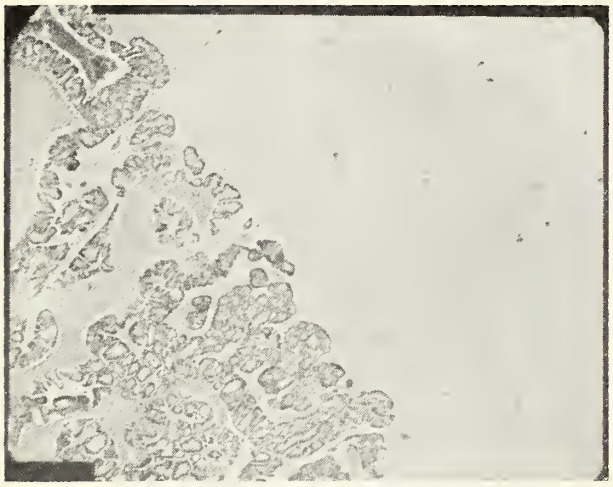


Figure 2. (H&E Stain × 25) Glandular pattern of the adenocarcinoma of the colon with rim of normal spinal cord tissue.

metastases to the vertebrae and extradural spaces. Elsberg[2] reports 254 cases of spinal cord involvement by tumor, 67 of which were metastatic. All of the latter were extradural. Wilson and Rupp[3] listed 53 cases of metastatic involvement of the spinal cord, two of which were intramedullary metastases. Baron and associates[4] reviewed 127 cases of spinal cord involvement by metastatic disease and found only two with primary metastases to the substance of the cord. In Benson's series, one case of carcinoma of the colon metastasized to the upper spinal cord at the level of T-4 as mentioned by Sansoy[5] in 1955.

Suggested routes by which the tumor may spread to the substance of the spinal cord include: (1) the circulatory system; (2) the lymphatic system; (3) the vertebral vein system; (4) embolic spread through subarachnoid spaces; and (5) direct involvement of the substance of the cord by extradural or dural tumors.[6] It is theoretically possible for tumor cells to reach the spinal cord by any of the first four methods and there are possible examples of each of these methods in the literature. At present there is insufficient evidence to select any one of these as the most common.

Why is intramedullary metastasis so rare in comparison to extradural metastasis? The cord has a rich vascular supply with many intercommunicating vessels. Most of these are small, however, and may be less acceptable to hematogenous spread. There is also a paucity of lymphatic channels in the substance of the cord. The expanse and location of this metastasis indicate that surgical intervention would have been both hazardous and fruitless. External radiation therapy directed to the metastasis temporarily reduces the tumor, but x-ray radiation to the spinal cord along with nitrogen mustard therapy was tried in two cases reported by Benson without evidence of success.

Summary

This patient experienced a cervical intramedullary metastatic neoplasm presenting itself as an acute hemiplegia and Brown-Sequard syndrome in the second week, quadriplegia in the third week, and death by the end of the fourth week.

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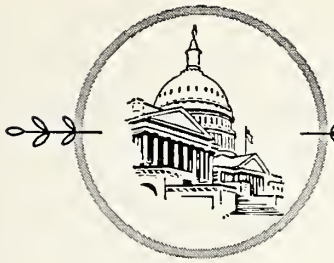
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Socio- ECONOMICS

YOU, the Telephone Manager

Ed. Note: This is the third in the series of articles prepared by the AMA Department of Practice Management, Division of Medical Practice. For other articles on this topic, see the following issues: November, 1977, and December, 1977.

The subject of practice management has become more critical in recent years. To assist the physician in a smoother operation of his office, the Department has produced workshops for medical assistants dealing with such aspects as collections, public relations-telephone techniques and office management, as well as practice management workshops for the new physicians. The Kansas Medical Society Executive Office will present such workshops upon demand by members.

THE RECEPTIONIST who answers the telephone in your office is "personality" to the dozens of patients who call each day. Some 90 per cent of all new patients phone an office and talk to her before ever being seen by you, the physician.

But your receptionist is not only important from a public relations standpoint; she can greatly affect the efficiency of your practice as well. For example, you could see more patients with fewer interruptions if your receptionist effectively screens calls, takes complete messages, and holds them for "call backs."

To do this effectively and without offending patients, your receptionist needs to be trained. Answering the telephone in your office is not as simple as answering calls at a retail establishment. And while few physicians delegate even the most minor of clinical tasks to untrained personnel, most physicians routinely assign telephone answering (which has the potential for an even greater impact on the practice) with little or no instruction, or follow-up.

Consider emergency calls, for example. It is important that your medical assistant-receptionist know what your concept of an emergency is, when such a call should be put through to you immediately, and what to tell a patient if you are not in the office when the call comes in.

Your receptionist should know how to communicate to patients calling in with a minor problem that you are concerned and will return the call. She should not brush patients off with, "Well, he's awfully busy right now and I couldn't interrupt him with such a small problem. I'll try to have him call you." All patients consider their problems to be important. Putting off patients hastily will usually mean repeated calls later, to ask if you are still busy. Such calls only increase the already heavy telephone load and tax the patience of your harried receptionist.

Handling emergency calls and "call backs" are only a few of the situations your receptionist should be able to handle. What about the "no-show" patient calling for another appointment? Should your receptionist mention the previously missed appointment and, if so, how should she bring up the topic? How about requests for medical information from insurance representatives and attorneys? Does she know who your friends are and what business associates to put through immediately? More than her common sense is required to deal with these situations. She must know what your common sense dictates and what the medico-legal implications of her decisions are.

The AMA realizes the importance of medical assistant and receptionist training in this area and has developed a course: "YOU, the Telephone Manager," which has been conducted for thousands of medical office personnel nationwide, in cooperation with numerous state and local medical societies. All of these situations and more are discussed in depth. One of the brochures used in the course is available to individual physicians, "Talking With Patients," which outlines some of the conversational "do's" and "don'ts" for receptionists to follow.

There are a few things you can do, too, to "tame the office telephone":

1. Don't just tell patients, "Call me anytime." Tell them to call, but explain that your office operates on a call-back system unless it is a real emergency and to cooperate by leaving a message;

2. Encourage your patients to communicate their medical problem to your assistant, so she can leave you a complete message and have their charts ready for you to consult when you return their call;

3. Return your calls! And try not to make call-backs at 5 PM. If the patient does have a serious problem that you feel needs attention, you will have to attend that problem at an inconvenient time for you, and your patient may have been needlessly suffering all day with a problem they called about at 10 AM.

You can try a call-back system most consultants recommend: Set aside several 10- or 15-minute call-back

(Continued on page 35)

Profile

The Physician's Assistant in Kansas

STEPHEN C. GLADHART, Ed.D.,* MERIDITH CRESPO† *and* V. GARY ANDERSON, M.D.,‡ *Wichita*

Results of a survey of Physician's Assistants who received their training in the Wichita State University Program, and are now working in Kansas, are presented and discussed.

The Wichita State University Physician's Assistant Program is continually gathering data to assess the extent of employment and the scope of work for program graduates. The research attempts to provide a profile of the graduate Physician's Assistant (PA) by examining the employing physician, practice setting, and patient acceptance data.

The WSU/PA program was established in September 1972 for the purpose of training Type A Primary Care Physician's Assistants. Except for a small initial class of twelve, the program has continued to enroll 24 students per year. The program length is two years with a combination of academic and clinical experience. The American Medical Association has granted full accreditation for the educational program according to the guidelines of the *Essentials of an Approved Educational Program*.[1] Additional historical aspects of the WSU program have been previously detailed.[2]

Background for the Study

One of the major elements in any study of PAs or other allied health professional groups is what happens after graduation. It is not enough to say that the training program is educationally sound if the graduates of that program are not accepted in the medical community, do not fill an identified need, or are not properly utilized in a manner which respects their skill and knowledge level. The purpose of this study was to provide a profile of the WSU graduate PA, with particular attention to those working in Kansas.

The contract establishing the WSU/PA program contained five major goals as guidelines for program development. In order to determine whether these goals were being met, it became necessary to formulate an evaluation procedure which would measure variables affecting the selection of students, the effectiveness of the program curriculum, and graduate employment data.

In order to assess the impact of the WSU/PA program on health care in Kansas, a comprehensive evaluation plan was formulated. The plan is comprehensive in that input (student selection), process (the teaching program), and output (graduate information) data are included. Graduate information is the major focus of this report.

Research Questions

Analysis of the program goals and informal observation led to the development of the following research questions regarding WSU graduates:

1. What are the demographic factors affecting the employment of PAs in Kansas? Are PAs employed in urban or rural areas? In what types of practice settings are PAs working? Are PA age and sex significant variables in employment?

2. Does the rural or urban background of the student affect the employment pattern of graduate PAs? Are Kansas residents more likely to choose rural areas after graduation than non-residents?

3. How does the graduate PA perceive his/her role in the patient-care setting? Does he/she relate well to patients? Has the PA established harmonious working relationships with physicians and other health personnel?

Methodology

A graduate survey questionnaire was constructed to measure employment data as perceived by the graduate PA. The survey was constructed following a study of the evaluative efforts of other allied health programs

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with items developed to answer questions unique to the Kansas program. The survey is intended to coordinate with other research regarding PAs in the state.[3]

The population for the study consisted of 47 graduates of the WSU/PA program; 33 males and 14 females are included in the analysis. Program graduates are surveyed at least six months after their initial employment since some questions refer to conditions which change over a period of time.

The questionnaire measures the PAs *perception* of his/her own performance and working relationships. Generally, the survey attempts to gain an overall impression of the employment pattern of WSU/PA graduates and to identify characteristics or variables which affect employment and utilization of the PA in the work setting.

The survey contains 17 separate variables. Arranged in grid fashion, 136 comparisons are possible between variables without repetition. Thirty-eight comparisons were selected which emphasize important factors in the employer-employee relationship. Descriptive data, charts, and graphs are used to present the most significant findings.

Results

Analysis by Geographic Area

The employment pattern for WSU/PA graduates indicates the acceptance of the concept and the availability of employment in Kansas. Over 80 per cent of the graduates have taken positions in the state with the large majority settling in Wichita and Western Kansas. Wichita is currently the only urban area in Kansas employing PAs trained in the WSU program, possibly because of local physicians' familiarity with the training program.

PAs have been accepted in rural and small town areas *(Table I)*. Of those graduates remaining in Kansas, 74 per cent have accepted positions in small towns or rural areas. A majority of these PAs are employed in communities of less than 25,000 population. Two major factors may be identified in the rural employment pattern of PAs in the state. First, other literature indicates the need, and consequently high demand, may exist in rural Kansas. A second influence may be the design of the training program which devotes a large portion of the clinical year to teaching rotations with physicians in rural areas of the state. It is quite probable that both factors have influenced the distribution pattern.

Although a demand may exist, the PA must be willing to relocate and work in a rural area. The variables of PA age and sex were examined to determine whether these characteristics have influenced the pattern of urban-rural employment. The age of the PA did not differ significantly between community categories. However, the difference between male and female employment patterns was significant.

TABLE I

GRADUATE PA's EMPLOYED IN URBAN AND RURAL AREAS

Size of Community	%	*N*
Less than 2,000	17.02	8
2,000-5,000	36.17	17
5,000-10,000	8.51	4
10,000-25,000	10.64	5
25,000-50,000	2.13	1
50,000-100,000	—	—
Over 100,000	25.53	12

Male PAs are much more likely to settle in less populated areas. Only 17 per cent of the male graduates are employed in areas of over 100,000 population, while 83 per cent of the male graduates are working in communities of less than 25,000. On the other hand, 75 per cent of the female graduates are employed in metropolitan areas. Although the female sample in this study is small (N = 14), the data indicates a significant difference in the orientation of male and female PAs in job preference. Family obligations are most often cited as reasons for low female mobility. Female PAs do, however, provide the state with a greater return on the training investment with 90 per cent remaining in the state after graduation, while 25 per cent of males leave Kansas after training.

PAs in rural Kansas are employed by physicians who are slightly younger than their urban colleagues. The average age of physicians employing PAs in Kansas is 44 years. Although not a significant difference, the average age of employing physicians in areas of less than 100,000 population is 43 years, while the age of physicians employing PAs in the urban setting is 46 years. This data is counter to the findings of other PA program research which has generally found rural employing physicians to be slightly older.

The Employment Setting

Since the WSU/PA program emphasis is on primary care, the employment of graduates in family practice and general practice settings is to be expected. The percentages of male and female PAs working in specialty areas are identified in *Table II*. The large percentage of female PAs in internal medicine reflects the

TABLE II

SPECIALTY OF EMPLOYING PHYSICIANS FOR MALE AND FEMALE GRADUATE PA's

	PA Sex					
	MALE		FEMALE		TOTAL	
Physician Specialty	N	%	N	%	N	%
Family practice	27	81.8	9	64.3	36	76.6
Internal medicine	1	3.0	4	28.6	5	10.6
Surgery	3	9.1	—	—	3	6.4
Other	2	6.1	1	7.1	3	6.4

employment of females in institutional settings. However, female graduates are finding greater opportunity in family practice as evidenced by continuing increases in that category. Compared with data gathered last year, fewer graduates are working in specialty areas.

The practice setting of physicians employing PAs is represented in *Table III*. The majority of PAs are working with physicians in solo and partnership settings. This is consistent with the fact that most PAs are employed in rural areas, where solo and partnership practices are more common. Supervision of the PA by a physician is a more complex issue in the institutional setting and consequently Federal institutions account for the seven graduates indicated in the data.

Contracts

Contracts between physician and PA, delineating the terms of employment, have been suggested in the literature. Our study indicates that PAs in specialty areas are more likely to have written contracts with their employer than are those working in general or family practice.

TABLE III

PRACTICE SETTING FOR MALE AND FEMALE PA's

	PA Sex					
	MALE		FEMALE		TOTAL	
Practice Setting	N	%	N	%	N	%
Solo	15	45.5	4	28.6	19	40.4
Partnership	5	15.2	6	42.9	11	23.4
Group—single specialty	4	12.1	2	14.3	6	12.8
Group—multi-specialty	4	12.1	—	—	4	8.5
Institutional	5	15.2	2	14.3	7	14.9

For those who have written contracts, the following provisions were most often reported: (1) salary; (2) insurance; (3) vacation; (4) salary increases; and (5) relationship to physician. Although less frequent, 12 PAs did report that a definition of tasks was included in the contract.

Factors Affecting Rural and Urban Employment

The maldistribution of health services has been discussed in many studies of the health care system. Of course, in Kansas the major concern has been with the differences between rural and urban areas of the state. The utilization of PAs has been proposed as a method for relieving the pressure on physicians in underserved areas. The federal contract establishing the program indicated that the faculty should consider the employment of PAs in underserved areas as a major goal. It would be difficult to assess all of the factors which might influence a graduate PA to seek employment in the rural environment. However, two such factors, resident status and previous geographic background were examined.

It was hypothesized that graduates who have been previous residents of Kansas would be more likely to accept a position in the state's rural areas. The results of the survey, as presented in *Table IV*, indicate slight differences between residents and nonresidents.

The differences are not great enough to determine employment patterns on this factor alone. However, when the rural and urban background of the PA prior to his/her training was studied, the results were significantly different. *Table V* indicates that the background of the student prior to entering training can be a primary factor in the graduate's decision to seek employment in a rural or urban geographic setting.

PA Acceptance

The PAs surveyed reported good acceptance from patients, physicians, and other health personnel. It should be pointed out that this data represents the

TABLE IV

THE RELATIONSHIP BETWEEN THE RESIDENT STATUS OF PA GRADUATES AND EMPLOYMENT IN URBAN AND RURAL AREAS

	Graduate Employment		
PA Background	URBAN	RURAL	TOTAL N
State resident	(19) 31.7%	(41) 68.3%	60
Non-resident	(4) 40.0%	(6) 60.0%	10
Total N	23	47	70

TABLE V

THE RELATIONSHIP BETWEEN THE GEOGRAPHIC BACKGROUND OF PA GRADUATES AND EMPLOYMENT IN URBAN AND RURAL AREAS

	Graduate Employment		
PA Background	URBAN	RURAL	TOTAL N
Urban	(18) 41.9%	(25) 58.1%	43
Rural	(5) 19.2%	(21) 80.8%	26
Total N	23	46	69

PAs' own feelings of acceptance or rejection.

Overall, PAs feel that patients have accepted their service, although in some cases, the patient may express hesitation upon first contact. Fifty-five per cent of the graduate PAs report that they have never had a patient refuse their service. Many have had experiences with patients who expressed acceptance following initial hesitancy. A large majority have patients who request their services. This reflects the degree to which PAs have been integrated into the practice of the employing physician. Taken together, the data indicate that the PA feels he/she has established good patient rapport.

Patient acceptance was also researched from the standpoint of PA age and sex. Perceived patient acceptance varied little according to age, but differences do exist between male and female PAs. Males report higher levels of patient acceptance than female PAs. There may be several explanations for these results. The data may be a reflection of the small number of female PAs and their employment conditions. Another factor may be the sensitivity of the female to the attitudes and feelings of patients. The female PA may feel the pressure of being a practitioner in an area normally held by males. This pressure, real or perceived, may create role conflict for the female PA which is in turn reflected in the patient acceptance data.

Good working relationships with physicians and other health personnel were universally reported. All PAs reported excellent working relationships with their employing physicians and office nurses. Work relationships with nurses outside the office were described as good in all but one case. Ratings for hospital administrations, other physicians, and other health personnel were all above average.

Conclusions

The results indicate that the original goals and objectives of the WSU/PA program are being achieved. A majority of the graduates have remained in Kansas and most have been employed in small town or rural areas. Employment has primarily been in solo, partnership, and single specialty group settings.

Overall, PAs report good patient and co-worker relationships. Further study should attempt to examine the acceptability of PAs from the perspective of employing physicians and patients. The results indicate the need to educate patients about the role of the PA in the office.

The employment patterns and perceptions of male and female PAs differ. More research, with larger sample populations, could identify the extent of these differences. Employers should be open to possible role conflicts in both male and female PAs. The occupation is in its infancy, and a lack of knowledge may result in misunderstandings. The employing physician should clearly understand the proper utilization of the PA and delineate the position for other health personnel. Role conflict will be reduced with increased definition and understanding of the concept and role of the PA in the health care system.

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Elements of Decision

Why Physicians Practice Where They Do: A Review

ADIB F. FARHA* ***and*** **DEAN KORTGE,† Ph.D.,** ***Wichita***

STATES ASPIRING to provide adequate and readily accessible health care for their populace must have a proper understanding of factors that motivate physicians in their location decision process. The local role played by physicians in the delivery of this care can hardly be overstated. Furthermore, an acknowledged link exists between physician's motivation, and services and appropriate health care.

This paper will explore, through a review of the literature, the factors underlying physicians' motivations and behavioral patterns as they relate to their initial career choice and to their location. With this as a baseline background, an attempt will be made to suggest guidelines for activities directed at physician recruitment.

Who Goes Into Medicine?

Young people entering the medical field are affected by much more than the promise of sizeable monetary returns. Pecuniary considerations are supplemented by several others—the character of the work in different occupations, the responsibilities involved, the possibilities of rendering service, their social standing, the hardships and pleasures attached to work, and so on.[1]

Additionally, financial problems are rarely, if ever, a barrier from entry to medical schools.[2] Several programs, on both the federal and state levels, exist to aid medical students in financing their medical education. In return for such aid, a number of these programs require some sort of commitment from the recipient to locate his/her practice in a certain geographical area or to enter a certain specialty. One such program operating in Kansas is the Kansas Medical Service Project, Inc. (Mediserve). Besides its lobbying efforts on behalf of students who, by virtue of background and experience, are especially motivated for rural medical practice, Mediserve provides loans on a need priority basis to cover tuition. Other examples of similar projects are numerous, such as individual community assistance plans, armed services programs, etc.

With minor exceptions, such as the draft of physicians at times of war, the government's position on the individual physician's decision-making process has been essentially one of non-intervention. Yet policies have lately been enacted that, although not interfering directly with such decisions, provide attractive incentives that strive to put the physicians' behavioral decisions in harmony with national policy guidelines. Nevertheless, state and federal guidelines cannot replace active involvement by communities, based on a good grasp of physician motivators and behavioral patterns. In the following sections, this paper will summarize research findings and look into their applicability to the recruitment efforts of concerned Kansans.

Factors which motivate physicians in choosing a practice locale are presented and discussed.

Behavioral Patterns of Recent Medical Graduates

A recent study[3] established the following six categories of factors indicated by previous researchers to be likely to affect physicians' practice location:

1. *Previous Attachment to the State:* Assuring the retention of an adequate supply of physicians in the state has been a major underlying rationale for public support of medical education. Men and women who receive their medical education in a state and continue through their internship and residency in it will likely develop contacts and attachments that make it more attractive to practice in that state. Hence, such people should be a primary target of recruitment efforts.

2. *Barriers to Entry:* Licensure standards of different states have an effect on location decision. However, such effect is minimal to the physician population in general and is more important to the general practitioner. A proper understanding of the State licensure standards is, therefore, basic to successful recruiting. Furthermore, administrators should work actively, in cooperation with their medical staffs, to secure significant input in reviewing licensure standards and hospital staff privileges which will not prohibit or dis-

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courage physicians from locating in their state and hospital.

3. *Opportunities for Professional Development:* Research findings indicate that the presence of a medical school in an area attracts specialists but has had a reverse effect on general practitioners. Therefore, communities located away from the state medical school could utilize this fact to attract general practitioners, who are the most urgently needed in most areas.

4. *General Environmental Conditions:* The degree of urbanization, as reflected by the number of people living in the state's urban areas, has a positive influence on location patterns. Additionally, the types and availability of recreational opportunities significantly affect physician location choices. Fortunately, this factor primarily influences specialists' location and its effect is hardly significant for those in general practice. Nevertheless, during recruitment activities, recruiters should stress the short time-distance of their community to the nearest resort center to offset the lack of recreational facilities in their own community.

5. *Effort:* With other things being equal, physicians will be more inclined to settle in areas where a given level of income can be achieved with the least number of hours in the office. Because of increased economics, division of labor, increased productivity, and so on, group practices offer physicians the promise of higher income with less effort. Where it is feasible to support more than one physician in a community, the possibility of setting up a group practice will enhance the possibility of attracting young physicians to that community.

6. *Income:* The new physician's income is decided by: (1) the present mean income of the state's physicians; (2) the probable relationship of his/her income to the mean; and (3) the possibilities and magnitude of expected changes in the mean. A good working knowledge of these factors is essential to successful recruiting.

Incorporating the preceding six categories into a sophisticated model, the findings placed the heaviest emphasis on the effects of previous contacts or ties with the given state prior to his/her first location decision. Looking at the four relevant events in the physician's professional life (birth, medical school, internship, and residency), it was found that the more contact a physician has with a state prior to the first practice location decision, the more likely he is to locate in the state. In other words, the probability of a physician who has had all four of these events in Kansas to locate in Kansas is higher than the probability of another physician who has had three such events in Kansas, and so on.

Furthermore, it was found that more recent events have a more impressive impact than earlier ones.[4–6] Hence, where a physician takes a residency is more influential on the location decision than where he took his internship, which in turn is more important than where he had his medical education.

Another major finding is that the effect of other factors (income, environment, effort) are less significant when the number of previous contacts is recent and extensive and have a strong impact only when such contacts are few and in the distant past. Recruiters should be aware of these facts and thus concentrate their efforts on physicians who have had the greatest number of contacts with Kansas. Those who had their residency in Kansas hospitals are more likely to practice in Kansas than those who went to medical school here and served their residency elsewhere. However, those who had both of these events in Kansas are even more likely to stay than any others.

Environmental conditions, which can hardly be changed through legislative mandates or by community groups, can be compensated for with special subsidies to encourage physicians to settle in less attractive areas. It would be logical for the impetus to develop the resources for such incentives to come through hospital administrators.

Rural or Urban Practice

The results of another survey revealed a wide array of personal and professional considerations that physicians listed as factors in their location decision *(Table 1)*.[7] Interestingly, no one factor was ranked by more than 50 per cent of the respondents to this questionnaire. However, among the factors listed, the opportunity to join a partnership or group practice was the one cited by the largest percentage. This factor seems to be taking an increasingly significant role, reflecting a national trend of physicians away from solo practice and into group arrangements.[8, 9]

Climate or geographical features of the area was the second most cited criterion. The next two factors do suggest policy programs, namely the availability of clinical support facilities and the opportunity for regular contact with a medical school or medical center. The implications of these findings reflect the significance of developing ties with medical centers and medical schools and of emphasis to potential recruits of the opportunities for continuing medical education and for participation in the outreach programs of the medical school.[10]

Further research examining the extent to which these factors overlap as explanations of practice location

TABLE I

FREQUENCY OF FACTORS RANKED FIRST, SECOND, OR THIRD BY ALL PRIMARY CARE PHYSICIANS

Factor	*Per Cent*
Opportunity to join a desirable partnership or group practice	43.0
Climate or geographic features of area	34.6
Availability of clinical support facilities and personnel	21.6
Preference for urban or rural living	21.5
Income potential	16.5
Opportunity for regular contact with a medical school or medical center	15.9
Influence of wife or husband (her/his desire, career, etc.)	15.6
Having been brought up in such a community	14.0
Having gone through medical school, internship, residency, or military service near area	12.3
Recreational and sports facilities	12.0
High medical need in area	11.7
Quality of educational system for children	10.3
Opportunity for regular contact with other physicians	9.7
Influence of family or friends	9.2
Access to continuing education	8.9
Cultural advantages	7.8
Opportunity to work with specific institution	5.9
Opportunities for social life	3.5
Prosperity of community	2.6
Organized efforts of community to recruit physicians	1.9
Advice of older physician	1.8
Prospect of being more influential in community affairs	1.7
Influence of preceptorship program	1.0
Payment of forgiveness loan	1.0
Availability of good social service, welfare, or home care services	1.0
Availability of loans for beginning practice	0.8

shows the payment of forgiveness loans as having the highest impact. Other factors, in order of priorities were: (1) organized efforts of communities to recruit physicians; (2) medical school contact; (3) social life; (4) place of rearing; and (5) influence of preceptorship program.[11]

A survey of physicians' wives indicated that 81 per cent felt that they had little or no influence on their husband's decisions. Similarly, 84 per cent of the physicians indicated that the wife's opinion did not significantly affect such decision. However, surveying the remaining 19 per cent of the wives, it was found that the physician would be more likely to practice in an urban area if his wife felt that it would increase the opportunities of her own career. On the other hand, a spouse was more supportive of a rural practice if he/she felt that lifestyle (urban or rural) was important to the physician, and if the availability of adequate social service, welfare, or home care services was highly rated on his list of factors.[7]

The implications of these findings are that rural communities that strive to attract more physicians should seriously consider developing good social services and home care services, and opportunities for the wives' careers. This might include employment opportunities and provision of acceptable day care for children.

A handy clue to the thinking mechanism operating in the minds of recent medical graduates is the following list of "Things to Consider When Shopping for Your Practice Location." This list was developed at the Bowman Gray School of Medicine as part of a simplified rating system that is suggested for use by entrants to the field in deciding on their practice location *(Table II)*.[12]

Summary

The importance of previous ties to the state, most pronounced through the residency years, cannot be overemphasized. Hence, those years are a good time for communities to contact physicians whom they hope to attract. Emphasizing the residency period does not negate the importance of the years in basic medical education. As pointed out earlier, if the physician had both his residency and his basic medical education in the state, he is even more likely to practice there.

The role of the spouse should not be overlooked. Providing the social setting and the school system that is attractive to the spouse is essential. It is also important for communities to develop ties with nearby medical centers and to have adequate support facilities. Most physicians would find it boring to practice in an area that does not have a hospital. They resent having their practice limited to the treatment of common colds and such minor ailments while missing out on the "interesting" cases that, by necessity, have to be referred to a town with a hospital and, hence, be taken care of by another physician.

Group practice is gaining increasing significance. Fear of professional isolation, fear of malpractice, the desirability of consulting on a regular basis with one's peers, and so on, are pushing physicians in this direction. Recruiters should, where possible, try to arrange for group practice arrangements when trying to attract new physicians.

It is crucial that communities and health care provid-

TABLE II[12]

THINGS TO CONSIDER WHEN SHOPPING FOR YOUR PRACTICE LOCATION

1. Availability of adequate hospital facilities.
2. Acceptability of location to spouse.
3. Availability of practice coverage at times.
4. Openness and receptivity of medical community.
5. Quality of interpersonal relations with other physicians in area.
6. Quality of school system.
7. Adequacy of support services such as laboratory.
8. Availability of consultation with specialists.
9. Geographic location.
10. Similarity of physician's background and the community.
11. Availability of adequate patient population.
12. Availability of recreational and cultural activity.
13. Availability of excellent continuing education.
14. Population size of the community.
15. Availability of office space.
16. Indices of economic health of the community.
17. Progressiveness of local hospital administrator.
18. Full-time emergency room coverage by others.
19. Possibility of regular work hours.
20. Potential annual income.
21. Medico-legal environment of the community.
22. Solo practice as prevailing mode of practice.
23. Previous familiarity with area.
24. Family ties (or lack of) to area.
25. Opportunity to join a desirable partnership or group practice.
26. Opportunity for regular contact with a medical school or center.
27. Organized effort of community to recruit a physician.
28. Prospect of being influential in community affairs.
29. Need for your services in the area.
30. Forgiveness of educational loan.

ers have a proper understanding of physician motivation and base pursuant recruitment activities on such an understanding.

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CERTIFIED MEDICAL ASSISTANTS

A total of 1,586 CMA certificates were earned by those taking the AAMA certification examinations in June 1977.

Of this total, 1,019 passed the basic Certification Examination, entitling them to use the initials CMA (Certified Medical Assistant) after their names. Specialty category certification was awarded to 567 candidates, with the following certificates earned: 173 Administrative (CMA-A); 372 Clinical (CMA-C); and 22 Pediatric (CMA-Ped).

The National Board of Medical Examiners is now working with the AAMA Certifying Board in designing next year's certification examinations.

Beginning in 1978, the examinations will be offered twice annually—in spring and winter—at more than 150 test centers across the country. The 1978 spring examinations, including the Basic Examination and the Administrative and Clinical Specialties, will be offered on June 2, 1978 (deadline: January 15, 1978). The 1978 winter offering, consisting of only the Basic Examination, will be administered January 26, 1979 (deadline: October 1, 1978).

The American Association of Medical Assistants, a national organization of more than 18,000 individuals employed by physicians in their offices, hospitals and other medical settings, offers these examinations as a service to both member and non-member medical assistants. Information may be obtained by writing the AAMA Certifying Board, One East Wacker Drive, Chicago, IL 60601.

The President's Message

In days gone by, the *Burgermeister* in one foreign country was selected by placing a louse in the middle of a table; the candidates placed their beards on the edge of the table and the one with whom the louse took up was elected *Burgermeister*.

We have come a long way, but the power of a single vote remains today.

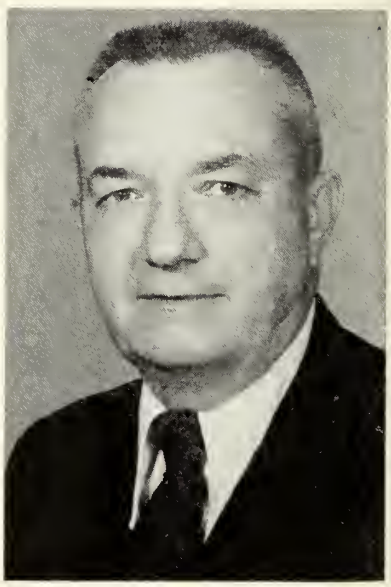
Thomas Jefferson was elected our third President by one vote in the electoral college. John Quincy Adams, our sixth President, was elected by one vote in the House of Representatives. Rutherford B. Hayes was elected President by one vote. His election, contested, was referred to a Congressional election committee. The matter was again decided by one vote. The gentleman who cast the deciding vote for President Hayes was also elected to Congress by a single vote. Andrew Johnson, our seventeenth President, was impeached by the House, but the Senate failed to convict him by one vote.

California, Idaho, Oregon, Texas, and Washington were admitted to the Union by a single vote each.

Numerous mayors, councilmen, legislators, and other public officials have been elected or defeated by a single vote.

To have any voice in your future, you must be politically active. Analyze the candidates; get out and support those who represent your ideals as to what is best for medicine and the people.

Bill Roy has declared that he is a candidate for the Senate. On the other side of the ticket, Jan Meyers has declared that she also will run for Senator. Still more will undoubtedly declare later. Now is the time for you to talk to these candidates, find out their views, try to impart your feelings, and see how they receive your message. Support the candidates of your choice, but support them with both manpower and that necessary evil, the almighty dollar.



Fraternally yours,

John D. Huff M.D.

President



Editorial COMMENT

Pattern for Control

Some years ago, the teaching of history consisted of enumerating a series of dates along with names, places, or events which were presumed to entrance school children with the wonders of the past. The method had two results. First, it turned untold numbers of school children away from any interest in happenings prior to last weekend. Second, this episodic presentation implied that these events and these individuals had sprung fully developed from some limbo and, having established the date as necessary for children to memorize, returned to it. Essential as these historical punctuations were, the method largely ignored the substance and spirit of the true historical continuity. The Declaration of Independence, for example (and which was written on July 4, 1776, as any school child can tell you), has been endowed with social, political, and literary value by subsequent events, but the history is in the thoughts and attitudes and pressures which brought it into being.

It behooves us, then, to look behind the reflex actions by which we respond to the daily assaults for the circumstances which are the true expression of history.

All of which, in our obtuse way, brings us to consideration of the Summary Report of the National Commission on the Cost of Medical Care. We are by no means intending to bestow on that document the sanctity accorded the D. of I. It seems certain its phrases will never be incorporated into Fourth of July addresses (though it may inspire some fireworks on its own). For one thing, it is written in what might be called Contemporary Committee Vernacular and Bartlett isn't likely to get out a new edition to accommodate it. Admittedly, it examines complex issues and in a prospective manner, which inevitably produces a certain amount of gobbledegook. But as we implied in the beginning, we suspect any historical significance in this report will rest not so much in the document itself as in its reflection of history which is enveloping us in these discordant days.

The commission, fostered by the AMA and composed of representatives of medicine, labor, business, government (both elective and appointive) and academia, assumed the task of determining why costs of health care have risen so much and, in particular, recommending what to do about them. That such an effort as this was even called into being reflects the current tenor of history and seems to ask: Have we reached a critical point of social consciousness which tells us that we should pause in executing various systemic alterations in our society, and ask what the cost is and, moreover, can we meet it? Or are we in the throes of a major upheaval in human values and relationships of which this is only a frenetic episode as we change our concepts of effort-remuneration to accommodate the full repertoire of social programs?

At any rate, the results of the study have been released and, apparently, with unanimity since no officially dissenting voices are recorded. It has been reported, however, that not all committee members are in agreement with all of the report, and we can expect controversy to be its most predictable product.

This prospect of controversy, however, can be taken as a measure of the success, not the failure, of the effort since the report deliberately addresses controversial subjects as is necessary in sorting through various options toward the most effective and acceptable ones. The report comments but makes no decisions. It expresses opinions but gives no answers. And it compiles, under appropriate headings, some 48 recommendations which will, it hopes, resolve the complex problem of financing health care.

Controversy will inevitably follow since these recommendations encompass about everything remotely connected with health care expense except the impact of child-proof bottle caps on frustrated adults. And just as inevitably, the controversy will be greater within and from the medical community than any other since it is understandably the object, directly or indirectly, of all the recommendations.

Essentially, the committee has approached and sought to resolve the problem of changing the prime concern in the rendering of health care from the traditional professional service consideration to the cost consideration. The idea that health service is to be unquestioningly available, and that its provision is to be gauged by the intrinsic medical import, is being displaced by the idea that the type and extent of medical service is to be controlled by how much it will cost and how that cost will be met. This is not to say that professional considerations will be eliminated but rather that they will be shaped by the capacity to support them.

The committee proposes that in order to contain health care costs the prime effort must be toward "strengthening price consciousness"—that is, establishing in the minds of both patient and physician that medical care is economically measurable, medical sources are economically finite, and therefore health services must be economically justifiable. It cites the variety of plans (both proposed and in effect) which have been devised to meet these matters and recommends that steps be taken to test them for effectiveness. It looks at the prospect of public utility regulation to see how it fits the medical scene (and finds that it may). It calls for the extension of information regarding physician services, insurance alternatives, and various mechanisms for the control of the quality of both. It considers the problems of hospital function and control as related to patient, physician, and community. It considers ways in which the professional actions of individual physicians will be brought under regulation by the imposition of economic, logistic, and professional (read certification) controls. And with what seems a somewhat conscious irony, it promotes on the one hand the control of costs through competitive practices while at the same time citing the inhibitory role of the necessary regulations on that same competitive effort. All this —as the advertisements say—and much, much more including, last but not least, the encouragement of "healthful life styles."

But fundamentally the report is a challenge. One can read into its implementation a long and strong governmental arm which will control our professional lives (but all other lives as well). Or one can see it as a preliminary sketch—by no means a definitive blueprint—by which, properly executed, the health profession can work to resolve its problems while keeping a continuing restraint on that governmental arm.

To return to our original thought, the report is but one of those punctuation marks in the line of history. The controversy it arouses will be about features and specifics. The significance, however, lies in the area of social philosophy as much as medical. We have the prospect of the geographic and political unit which has melded its natural and human resources into the most productive and successful system ever accomplished. As this society has reached a productive and expansive peak, a large part of its effort has been directed toward extending programs of benefits to those in need of various forms of assistance. But as the definitions of need have been extended, the question is coming home—how much for how many for how long—and by whom?

If the report embodies the wealth of controversy we anticipate, it also embodies the factors which will have to be studied thoroughly and dispassionately if we are to resolve them. It seems worth keeping in mind the historical sweep which has brought us to this point as we attempt to extend in all directions and at all levels the means of being our brothers' keepers. Perhaps each age sees itself as the one of greatest moment since time began —certainly this one does. But we are surely approaching a time of decision as to which of very different roads we take. For what do we do when the brothers outnumber the keepers?—*D.E.G.*

Vox Dox

Vox Dox Editor:

Allow me to congratulate you on your most recent editorial commentary in the *Journal of the KMS* dealing with "Mediserve." Your trenchant analysis of the production/distribution equation for physicians in Kansas blows away a lot of fog and is very clear and concise and convincing. It is just too bad that the public cannot read it as well as physicians.

I have always been bemused by the complaints that there are few doctors in the sparsely-settled far western parts of Kansas. Where would we expect doctors to be except where there are people? After all, physicians are designed, one hopes, by both training and inclination to take care of people, not antelopes or wheat fields.

There is one small quibble I would have with your comments and that is where you say "Medical education is in short supply all over the country, and it is good to have a school whose prime interest is in state residence." Probably you mean by this that there are many more candidates for each existing medical school position than there ever have been in the past. But I think that both of us would agree that in fact we have increased our medical educating capacity to the point where we are on the verge of a physician excess which will create its own unique problems. The one good thing about it will be that such an excess will inevitably cause an influx of practitioners into any area where it is possible to set up a viable practice.

If Mediserve does nothing more than act as a holding action against punitive and ill-advised coercive legislation, while the surplus of physicians distributes itself, it will have been a success.

J. H. Ransom, M.D.
Topeka Allergy Clinic
Medical Plaza Bldg.
Topeka, KS 66604

Vox Dox Editor:

We found the results of George Burket's "Rural Manpower" study (November 1977 *Journal of the KMS)* to broadly and statistically confirm some of the experience we have had during the past two years in working with medical practices, hospitals and communities in Kansas, Missouri, and Iowa.

As with several previous articles in the *Journal* on the issue of rural health manpower, we are commending it to many of the smaller urban and rural areas that otherwise might seek to "rediscover the wheel," as Dr. Burket so aptly put it.

I would simply add, in more explicit terms than the report on Dr. Burket's survey, that we have found group practice settings to offer an organizational framework for resolving many of the difficulties implicit in the concerns noted in the lower portion of *Table I* in the article. A preponderance of solo practice models in rural areas, and a sometimes legitimate apprehension of the disadvantages of group practice, compounds the problem of developing new rural practices. However, on the basis of limited first hand success, we are optimistic that the problems can be overcome and are on their way to improvement.

Ronald L. Hammerle, D.Mn.
Director, Health Resources
P. O. Box 12621
Kansas City, MO 64116

KANSAS UNIVERSITY MEDICAL CENTER, KANSAS CITY AND WICHITA FACULTY VISITS TO KANSAS COMMUNITIES

In an effort to develop a person-to-person program to better acquaint the faculty of the Medical Centers in Kansas City and Wichita with the community physicians throughout the state of Kansas and vice versa, members of the Kansas Medical Society Auxiliary and their physician spouses will be inviting faculty members and spouses (and in some instances, families) into their homes for weekends. To assist in matching families, please complete and return the following form to:

Mrs. A. Dean Burnett
504 College
Halstead, Kansas 67056
316-835-2920

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Address ..... Zip .....
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Names of family who will participate:

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Children	*Ages*	*Interests*
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Possible dates for participation:

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Other comments:

Obituaries

IVAN ROY BURKET, M.D.

Dr. Ivan Roy Burket, 89, died December 8, 1977, in Ashland. He was born June 24, 1888, in Clark County.

Dr. Burket graduated from the University of Kansas and from Harvard University Medical School. Following a surgical residency in Boston and service with the U. S. Army, he returned to Ashland in 1919, where he practiced until his retirement in 1975.

FREDERICK G. DEITRICH, M.D.

Dr. Frederick G. Deitrich, 58, died December 9, 1977, in Topeka. He was born March 24, 1919, at Clay Center.

Dr. Deitrich was graduated from the University of Kansas School of Medicine in 1944, and served for a time with the Army Medical department. He practiced in Lakin and Abilene prior to moving to Topeka in 1975.

Survivors include his wife, a daughter, and two sons.

FLOYD E. DILLENBECK, M.D.

Dr. Floyd E. Dillenbeck, 77, died December 2, 1977, in Sun City, Arizona. He was born February 13, 1900, in El Dorado.

Dr. Dillenbeck was graduated from the University of Nebraska School of Medicine in 1929 and practiced in El Dorado until his retirement in 1967.

Survivors include his wife, Carol, a son and a daughter. Memorial funds have been established in his name in Sun City with the Heart Association and with the Sunshine Service.

CME Relicensure in Kansas

Reporting Date Is Near—July 1, 1978

From and after July 1, 1978, every licensee rendering health care service in the state is required to submit with his or her request for license renewal evidence of satisfactory completion of a program of continuing education.

The Board of Healing Arts will accept completion of the AMA Physicians Recognition Award or the CME certification programs of those specialty societies recognized as equivalent to the Physicians Recognition Award.

Upon completion of the CME requirement, the AMA or the specialty society will notify the Kansas Medical Society office. A certificate (see sample below) will be mailed to you prior to your licensure renewal date. Please retain one copy of the certificate for your files and forward one copy to the Board of Healing Arts with your license renewal form.

Any physician licensed after July 1, 1975, will have three years to complete 150 hours of continuing medical education for licensure renewal.

If an applicant for renewal will not render health care service in Kansas, the applicant shall submit with his or her request for license renewal an affidavit stating that he or she will not render such health care service during the term of the license.

The Board of Healing Arts may grant a waiver for a specified period not to exceed eight (8) months to a physician who has suffered an illness or accident making it impossible or extremely difficult to comply with the CME requirement.

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Constitutional Secretary

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KANSAS MEDICAL SOCIETY

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TOPEKA, KANSAS
Downtown Ramada Inn

Sunday, May 7—House of Delegates First Session, 9:00 AM—Downtown Ramada Inn

Monday, May 8—Sports Day, Topeka Country Club

Tuesday, May 9—Scientific Session, Downtown Ramada Inn, 8:00 AM-5:00 PM

Wednesday, May 10—House of Delegates Second Session, Downtown Ramada Inn, 8:30 AM

Make Your Reservations Now!

YOU, The Telephone Manager

(Continued from page 19)

periods throughout the day. Your receptionist can dial one patient and have them on the line while you are talking to another. Or, make a few calls after seeing patients in two or three examining rooms to break up the routine. And, your receptionist will be able to tell patients, "You can expect the doctor's call within the hour, will you still be at this number and will you please keep your line free?" The benefits here are obvious: you can eliminate some calls to endlessly ringing numbers with no one home, and calls to patients who are phoning friends and relatives about their aches and pains resulting in busy signals ad infinitum;

4. Have an unlisted number in the office—for your use only. You can use this line to make outgoing calls to patients, physicians, the hospital, etc.;

5. Be sure your telephone equipment is up to date. Patients may not tell you, but your receptionist probably receives repeated complaints of, "I have been trying to get through to you for the last hour. . . ." Most telephone companies will do a free "busy signal" study to determine if your office has enough incoming lines. They will be able to give you an exact count of how many busy signals come from your office number each day for a week. This study can take the guesswork out of ordering expensive equipment you do not need. By trying some of these suggestions you will begin to manage and control the office telephone—which now may be managing and controlling you.

(To order, "Talking With Patients" OP-450; $.30; send remittance to AMA Order and Handling Department, 533 N. Dearborn Street, Chicago, IL 60610.)

CLASSIFIED ADVERTISEMENTS

Mid-America Cancer Center Program seeking Associate Director for Cancer Outreach. Experience in clinical aspects of cancer, education, and community relations. Contact James T. Lowman, M.D., Director, Mid-America Cancer Center, KUMC, 39th and Rainbow, Kansas City, KS 66103.

HEALTH RESOURCES has immediate practice opportunities for general practice and family physicians, two physicians interested in geriatric medicine, and an ENT. Other primary care and surgical openings are available July 1. Please send CV in confidence to: Dr. Ronald Hammerle, Health Resources, Ltd., Box 12621, Kansas City, MO 64116.

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MITCHELL V. KAMINSKI, JR., M.D., St. Mary of Nazareth Hospital Center, Chicago, Illinois.
JAMES C. WARREN, M.D., Ph.D., Washington University School of Medicine, St. Louis, Missouri.

Program Topics Include:

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THOMAS F. FRAWLEY, M.D., St. Louis School of Medicine, St. Louis, Missouri.

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March 31-April 4, 1978

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"THE HIGH RISK PATIENT"

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Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.

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Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral—Adults:* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.;* severe states, 20 or 25 mg *t.i.d.* or *q.i.d. Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

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The JOURNAL of the KANSAS MEDICAL SOCIETY

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□ **Appropriate antibacterial therapy:** up to three days therapy with Azo Gantanol, then 11 days with Gantanol® (sulfamethoxazole), 0.5 Gm tablets.

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: In adults, urinary tract infections complicated by pain (primarily pyelonephritis, pyelitis and cystitis) due to susceptible organisms (usually *E. coli, Klebsiella-Aerobacter, Staphylococcus aureus, Proteus mirabilis* and, less frequently, *Proteus vulgaris*) in the absence of obstructive uropathy or foreign bodies.

***Note:* Carefully coordinate *in vitro* sulfonamide sensitivity tests with bacteriologic and clinical response; add aminobenzoic acid to follow-up culture media. The increasing frequency of resistant organisms limits the usefulness of antibacterials including sulfonamides.** Measure sulfonamide blood levels as variations may occur; 20 mg/100 ml should be maximum total level.

Contraindications: Children below age 12; sulfonamide hypersensitivity; pregnancy at term and during nursing period; because Azo Gantanol contains phenazopyridine hydrochloride it is contraindicated in glomerulonephritis, severe hepatitis, uremia, and pyelonephritis of pregnancy with G.I. disturbances.

Warnings: Safety during pregnancy not established. Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been reported and early clinical signs (sore throat, fever, pallor, purpura or jaundice) may indicate serious blood disorders. Frequent CBC and urinalysis with microscopic examination are recommended during sulfonamide therapy.

Precautions: Use cautiously in patients with impaired renal or hepatic function, severe allergy, bronchial asthma; in glucose-6-phosphate dehydrogenase-deficient individuals in whom dose-related hemolysis may occur. Maintain adequate fluid intake to prevent crystalluria and stone formation.

Adverse Reactions: *Blood dyscrasias* (agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia); *allergic reactions* (erythema multiforme, skin eruptions, Stevens-Johnson syndrome, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis); *G.I. reactions* (nausea, emesis, abdominal pains, hepatitis, diarrhea, anorexia, pancreatitis and stomatitis); *CNS reactions* (headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo and insomnia); *miscellaneous reactions* (drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon). Due to certain chemical similarities with some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia. Cross-sensitivity with these agents may exist.

Dosage: Azo Gantanol is intended for the acute, painful phase of urinary tract infections. *Usual adult dosage:* 2 Gm (4 tabs) initially, then 1 Gm (2 tabs) B.I.D. for up to 3 days. If pain persists, causes other than infection should be sought. After relief of pain has been obtained, continued treatment with Gantanol (sulfamethoxazole) may be considered.

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This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** When the combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome. Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium sparing action of triamterene is warranted. (See Box Warning.) Routine use of diuretics in healthy pregnant women is inappropriate; they are indicated in pregnancy only when edema is due to pathological causes.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

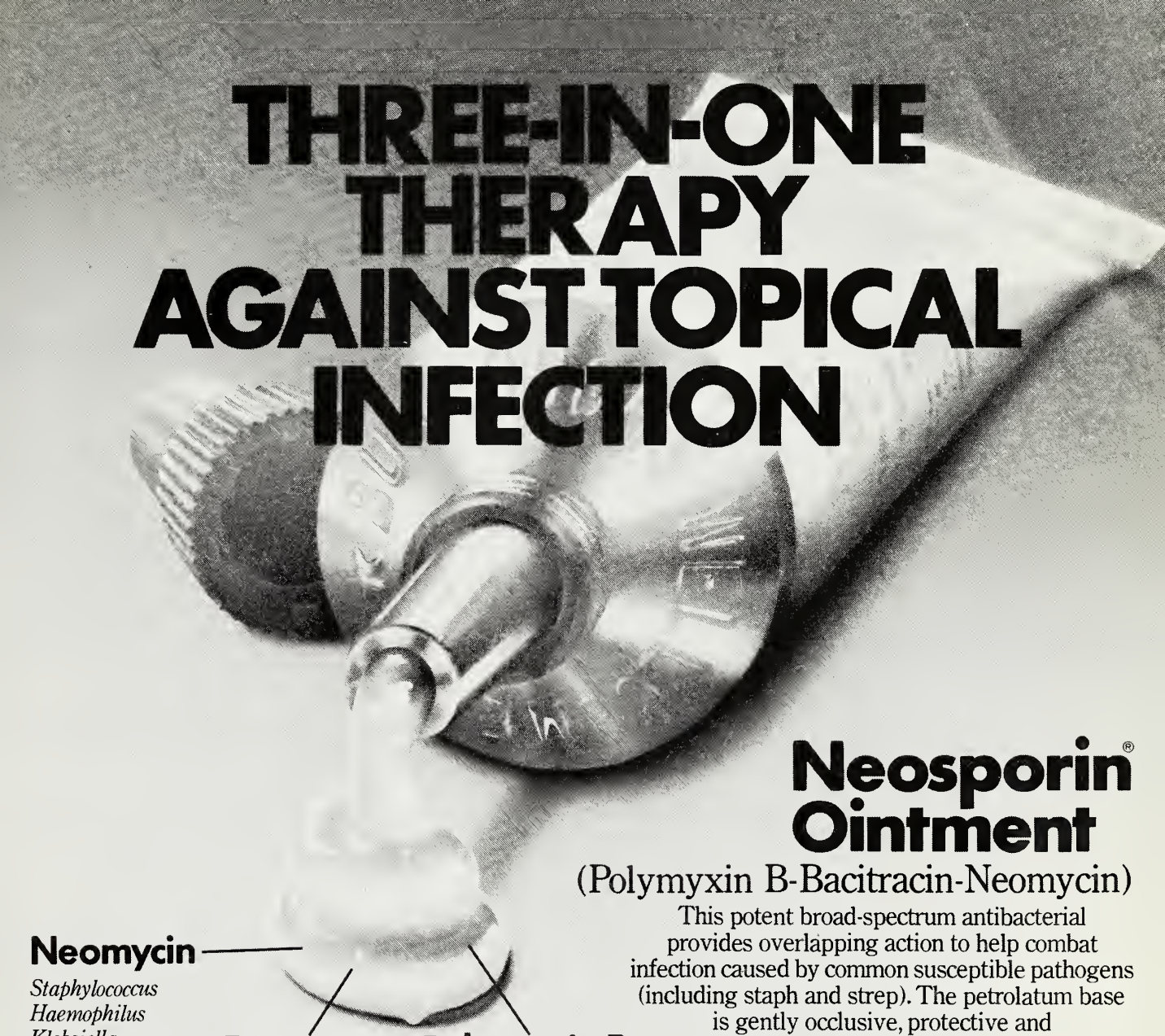
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This potent broad-spectrum antibacterial provides overlapping action to help combat infection caused by common susceptible pathogens (including staph and strep). The petrolatum base is gently occlusive, protective and enhances spreading.

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Streptococcus
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In vitro overlapping antibacterial action of Neosporin® Ointment (polymyxin B-bacitracin-neomycin).

Wellcome

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Neosporin® Ointment

(Polymyxin B-Bacitracin-Neomycin)

Each gram contains: Aerosporin® brand Polymyxin B Sulfate 5,000 units; zinc bacitracin 400 units; neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base), special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manitestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching, it may be manitest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken it this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

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WASHINGTON, Aug. 26, 1920—



Social Security Bill Is Signed; Gives Pensions to Aged, Jobles

Roosevelt Approves Message Intended to Benefit 30,000,0 Persons When States Adopt Cooperating Laws–He Call the Measure 'Cornerstone' of His Economic Program.

WASHINGTON, Aug. 14, 19
The Social Security Bill, provi
a broad program of unemployn
insurance and old age pensi
and counted upon to benefit s
20,000,000 persons, became law
day when it was signed by P
dent Roosevelt in the presenc
those chiefly responsible for
ting it through Co
Mr. Roosevelt cal
"the cor erstone
which is being
me s c ple
ri to

SENATE APPROVES 18-YEAR OLD VOTE IN ALL ELECTIONS

Amendment to Constitution is Sent to House, Where Passage is Expected

WASHINGTON, March 10, 1971—The Senate approved today, 94 to 0, and sent to

COMP

UL IGNE

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NEW WORLD HOPE

President Hails 'Great Instrument of Peace,' Insists It Be Used

HISTORIC LANDMARK

Meeting Gives Standing Ovation as Executive Pictures Peace Gain

"If we fail to use it," he declared to the solemn final meeting of the delegates, 'we shall betray all of those who have died in order that we might meet here in freedom and safety to create it.'

"If we seek to use it selfishly—for the advantage of any one nation or any small group of nations—we shall be equally guilty of that betrayal."

Fervent Interpolation

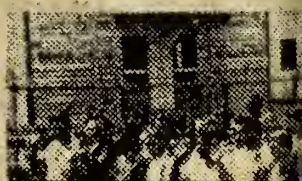
The President, speaking in the auditorium of the War Memorial Opera House, built in memory of sons of the Golden Gate city who gave their lives in the first World War, in which he himself served, seemed to give unconscious expression to the solemn feeling of the occasion when, at the outset of his speech, he interpolated the words, half a hope, half a prayer:

"Oh, what a great day this can be in history!"

Just before the plenary session

the Draft Ends Nov

WASHINGTON, Jan. 27, 1973—"With the signing of the peace agreement in Paris today, and after receiving a report from the Secretary of the Army that



PATIENT PACKAGE INSERTS: A CONCEPT WHOSE TIME HAS COME?

The consumer's right to know is an irreversible and desirable trend of the Seventies. It extends, and properly, to a patient's right to know more about his or her prescription medications. One way, gaining favor, is through patient package inserts. Wisely-prepared and properly distributed when medically indicated, they could markedly improve patient knowledge and drug therapy—laudable goals by anyone's standards.

The PMA endorses these goals and will work with government, the health professions and consumers to achieve them.

The Advantages

The concept holds promise of benefits: better patient understanding of the product prescribed, better adherence to the treatment plan, and more awareness of possible side reactions.

Every doctor has had patients who fail to finish antibiotic regimens because they feel better. Some patients assume that if one tranquilizer or analgesic is good, two may be twice as good. Still others fail to report dizziness while on antihypertensive therapy —and so on.

Problems like these might arise less often if the patient received written information in addition to verbal instructions. Some studies suggest that patients are more receptive to such materials, and they more often understand the verbal instructions and follow them, when inserts are used.

The Disadvantages

There are also some potential problems. Obviously, the inserts must be clearly phrased, without extraneous or complex detail. How much information is enough? How can it be kept current? Should all patients receive the same information? Should inserts be included with all drugs? Should only potential problems be listed or are patients better off with a "fair balance" presentation that describes usefulness as well as drawbacks?

These and similar questions require answers, since model inserts have yet to be properly developed and tested. Despite the need for these studies, the FDA is proceeding prematurely with inserts on selected products. We think the Congress is the only place where the matter can be given the proper legal status and direction, particularly since it represents a conceptual change in the legal, medical and social framework of the nation's prescription drug information system.

The Solution

The PMA believes that carefully-devised pilot studies of various kinds of inserts are needed. They should be developed and implemented with full participation by doctors, pharmacists, consumers, communications experts and the drug industry. Such studies will provide reliable pathways to follow, so that inserts will be useful aids to medical practice.

And particularly we think that you should be closely involved in this debate and in these studies and decisions. Otherwise, people with less experience and qualifications may control the purposes, content and use of a tool with considerable promise for improved patient care. It could make a difference in your practice tomorrow, and more importantly, in the health of your patients.

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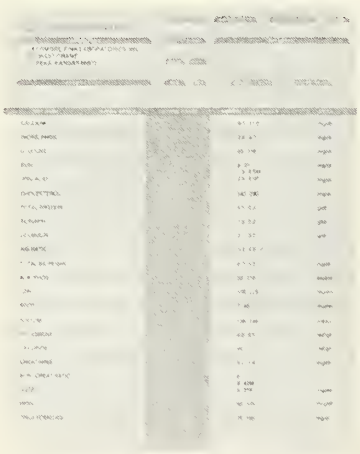
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Learning Disabilities: April 13 - 14, 1978. A multi-disciplinary faculty will address the national goal of comprehensive services for all handicapped children by 1980. Lawrence Bradford, Ph.D., will be the workshop leader.

CME credit available. All workshops will be in Topeka, Kansas.

For further information please contact June Housholder, Division of Continuing Education, The Menninger Foundation, P.O. Box 829, Topeka, Kansas 66601. 913/234-9566, ext. 3685.

30th annual MIDWEST CANCER CONFERENCE
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The Kansas Division of the American Cancer Society invites midwest physicians to attend a two-day graduate course on cancer, "Diagnosis and Management of Lung Cancer."

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Tumor Immunology
Fred Hutchinson Cancer Research Center
Seattle, Washington

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Oncologist
Audie L. Murphy Memorial Veterans Hospital
San Antonio, Texas

David R. Sanderson, M.D.
Thoracic Diseases & Internal Medicine
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Radiotherapy Department, M. D. Anderson Hospital & Tumor Institute
Texas Medical Center
Houston, Texas

AAFP and AMA Category I Physician Recognition Award credit. Registration fee—$10.00. No preregistration necessary. Exempt from registration: medical and nursing students, residents and paramedical personnel in training.



February is the month for giving valentines to very special people. I hope, Doctor, that you gave a big one to the spouse in your house. If she is like the many physicians' wives that I have met over the past ten years, it was certainly deserved. The other day I perused some old correspondence from past presidents of the Auxiliary who have patiently guided me in the organization. Some I have come to know well and others I know only slightly, but all are remarkable women.

Physicians' wives are as individualistic as their husbands. They are intelligent, talented, sensitive, and have strong value systems. I have tried to draw a word-picture, but find it difficult to construct a composite. These attractive and poised ladies can adapt to any crisis or demand. An Easterner's compliment was, "You Kansans are very friendly and down to earth, but always seem at ease in the most sophisticated situation, too."

The country-club-bridge-and-golf image that seems to dominate the public concept of the doctor's wife certainly doesn't fit the artists, musicians, equestrians, church elders, teachers, judge, Girl Scout and Campfire leaders, civic leaders, physicians, nurses, and volunteers that I know. Our public relations seem to have slipped as higher priorities are being given to volunteer service to hospitals, churches, youth groups, nursing homes, and other worthwhile causes. Increasingly, physicians' wives are returning to the job market after rearing their families or combining career and family responsibilities.

Most physicians' wives share a commitment to the goals and ideals of the medical profession to which they are wed. They are concerned about the problems facing the profession and are willing to help solve them if given encouragement and direction. Help us to help you; we really can make a difference.

Here's a big valentine and a special welcome to the men who are husbands of physicians; some day one of you will be writing this column.

From the 1300 plus members of the Auxiliary—we really do love you.

Sincerely,
Jackie Burnett
President
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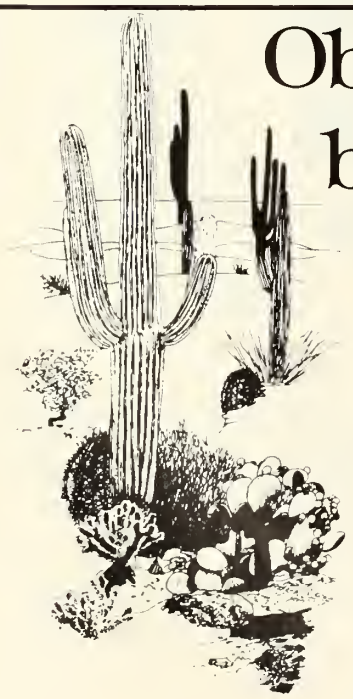
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HOW WE STAND THE COLD

Human beings are essentially semi-tropical animals. Our bodies at rest and unclothed are designed to maintain their internal temperature effortlessly with the thermometer at about 85 F. But built into us are effective methods of coping with much lower temperatures.

We achieve cold-weather comfort in part by generating more heat in our internal furnaces, and in part by conserving that heat.

The muscles, most important source of internal heat, use about 70 per cent of the food energy they consume, at work or at play, in heat generation. Under average conditions, body muscles produce enough heat to boil a quart of freezing-cold water every hour—and when you wave your arms or stomp your feet while waiting in the cold, you are stoking your muscle furnaces to a still higher level of heat production.

The extent to which muscular activity enables you to ward off the cold has been strikingly illustrated by the experiments of Canada's National Research Council, which show that the amount of clothing needed to keep you comfortably warm when sitting quietly at 70 F will also keep you warm at 40 F if walking briskly—or at –5 F if running.

If you don't ward off the cold by exercising voluntarily, your muscles take over and warm themselves by shivering involuntarily. Under extreme conditions of exposure, intense shivering may even save you from freezing to death. "It's largely shivering which explains why many are cold but few are frozen," one physiologist has said.

Since your muscles need to produce more heat in winter, they use up more food energy; but nature makes allowance for this by turning up your appetite a few notches in cold weather. On the average, you eat about 15 calories more per day for every one-degree drop in the temperature. Soldiers allowed to eat as much as they please when stationed in the tropics at 92 F selected a diet totaling about 3000 calories daily; in the Arctic or Antarctic at –25 F, their calorie intake rises spontaneously to nearly 5000.

Your tastes for particular foods also change during cold weather. Explorers report, for example, that men who carefully trim all fat off their meat back home crave and eat much of this prime source of heat on polar expeditions.

Instead of increasing your heat production in cold weather, you can achieve much the same result by conserving what heat there is. One simple method of heat conservation is familiar to everyone. When cold, you instinctively curl up into a ball, thus cutting much of the surface area through which your internal heat is dissipated.

Less familiar are your automatic blood and skin changes. Ordinarily, the blood and skin act as a cooling system like the water and radiator of your car. Hot blood emerging from the internal organs is cooled by flowing through the skin at the rate of 50 to 75 gallons/hour. When you are chilled, however, many small blood vessels in your skin close up, reducing the rate of flow to one fifth of normal, so that your body "turns pale with cold." The net effect of this bloodflow restriction is to convert your skin from a radiator for dissipating heat into a blanket for conserving it.

The efficiency of this skin blanket depends in part upon the thickness of the fat layer beneath it. In general, people with well-distributed fat deposits survive extreme cold better than their thinner fellows, which may explain why most successful channel swimmers have been generously upholstered. But fat people are not necessarily more comfortable in the cold; for the nerve endings which complain "I'm cold" to the brain are themselves located near the surface of the skin; they may actually end up colder than the thin man's—and sending out more insistent messages of complaint —if they are insulated from internal sources of heat by layers of fat.

These skin nerve endings, incidentally, appear to be superior in design to most similar devices developed by engineers and scientists. They are sensitive to sudden changes in temperature. When you step outdoors on a cold day they signal the change to your brain immediately, long before your skin has actually chilled to an uncomfortable level. And when you have been outdoors in the cold long enough so that heat production and heat loss are in balance, they send an "I'm comfortable" message to the brain even though their actual temperature has dropped off half a dozen degrees.

Conserving body heat depends in part on the materials with which your body or clothing makes contact. Thus the tile floor of your bathroom feels colder than the bathmat to your feet, even though both are the same temperature; heat flows more rapidly from your skin to a good heat conductor like tile.

Quiet air, fortunately, is a poor conductor of heat—much poorer than water, for example. The human body, which maintains its heat balance without effort in still air at 85 F, requires water at over 90 F for a similar balance. A man may die after 60 minutes in ice-cold water; he can live much longer in air at the same temperature. Wool socks and boots keep your feet warm at sub-zero temperatures while they are dry; but if water seeps in, your toes will soon start to numb. The

mother who steps outside for a minute to determine how cold it is, then bundles five-year-old Billy up in layers of wool before sending him out to play, is not being a thoughtful, cautious mother. She has forgotten that outdoor air feels much colder than it is when you first step into it. And she has forgotten that Billy is going to run and jump, thus increasing his internal heat production many times over. As a result, Billy is soon perspiring in his heavy togs. He sits down to rest. His internal heat production falls while his heat loss increases due to the dampness of his clothing. Soon Billy comes home with teeth-a-chatter, chilled to the bone. "I should have put an extra sweater on him," his mother tells herself.

A wiser mother sends a child out for strenuous play in relatively light clothing, but with adequate protection for his hands and feet; and she will remind him to come in for something warmer if he begins to feel cold.

Still air is an excellent insulating material, but moving air quickly carries heat away with it. Even a breeze blowing at 5 mi/hr carries away about eight times as much body heat as still air. The winter uniform of a soldier, for example, loses about a quarter of its insulating efficiency when he is walking briskly rather than standing, because breezes are generated within his clothing which carry off heat.

The loosely tailored sealskin and walrus-skin clothing of the Eskimos is nearly ideal for cold weather. When an Eskimo chases his quarry in a hunt, the chill Arctic air flows into and out of his flapping garments to prevent overheating. Later, when he sits down to rest, his clothing settles around him and achieves an insulating efficiency hard to surpass.

Most of us think of wool as ideal for heat conservation, and scientific studies have confirmed its excellence. The insulating effect is not achieved by the fibers themselves but by the air trapped among the fibers. Wool's superiority to cotton in this respect is due largely to its springiness. Damp or dry, it tends to regain its thickness more readily after compression, and to trap some more air. The suitability of some synthetic fibers for cold-weather use depends similarly on their ability to spring back into shape when compressed by bodily movements.

Fur also insulates by means of the dead air trapped among the hairs; and many animals are able to regulate their heat balance by expanding or compressing their fur. They achieve this effect by means of tiny muscles which erect the hairs and thus thicken the furry layer when the animal begins to chill. We human beings, although we lack fur, still have the same hair-erecting muscles in our skins; and these muscles still contract when we are suddenly chilled, producing goose pimples.

Understanding of the importance of thickness in clothing has enabled scientists to improve gloves for Arctic wear. Our fingers are partially curved most of the time, yet glove manufacturers tailor gloves to fit the fully extended hand. As a result, ordinary gloves are compressed to a fraction of their normal thickness at the joints and knuckles where are fingers bend—and heat leaks out. Military gloves, to minimize the compression, are now shaped to the natural curve of the relaxed fingers.

Keeping warm while asleep offers a particular challenge, for internal heat production falls during sleep. No doubt you have more than once had the experience of falling asleep in a comfortably heated room and awakening to find that you are cold and shivering. The room has not chilled while you slept; instead, your heat production has dropped. It is therefore wise to cover yourself with something when you lie down to take a nap even though you are sure you won't need it.

Electric blankets may seem like an exception to the general rule that bedding and clothing are designed to conserve your own internally generated heat; an electric blanket feels as if it were actually sending heat to your skin. But this is an illusion; such a blanket rarely reaches the temperature of your skin. Like any ordinary blanket, what it really accomplishes is to slow down the loss of your internal heat. The chief advantages of the electric blanket are three: (1) it warms itself so that you do not have to curl up and shiver in order to warm it initially from your own inner heat resources; (2) it provides a maximum of heat conservation with a minimum of weight; and (3) a good electric blanket adjusts itself automatically to changes in outer temperature, so that you do not have to add a blanket or take one off as the air around the bed chills or warms.

How much cold can the human body survive? Dorothy Mae Stevens was found unconscious, almost unclothed in a Chicago alley one winter morning in 1951. She had apparently lain there all night and her internal temperature had fallen to 64.4 F—34 degrees below normal. The hospital to which Ms Stevens was taken saved her life through good nursing care and the use of stimulants, blood plasma, oxygen, antibiotics, anti-blood-clotting drugs, pressure bandages, and other medical methods. Even more astounding was the 1955 case of two-year-old Vickie Davis, who survived after being found unconscious in her night clothes with an internal temperature of 60 F.

Naturally the adverse effects of exposure to severe cold when the body and clothing are dry are less severe

than when wet. It makes a big difference.

All water does not freeze at the same temperature. Ordinary fresh water freezes at 32 F, while ocean water, which is salty, freezes at 27.9 F. An article in the *British Medical Journal* stated that a sailor falling into the Arctic Ocean had no chance of survival.

If caught in sub-zero temperatures and upon reaching shelter chilled to the bone with nipped fingers, toes, cheeks, nose or ears, the ancient suggestion to rub the frost-bitten parts with snow or ice should be avoided. Recent research has shown that the immediate application of gentle warmth leaves less tissue damage and less likelihood of infection or gangrene. The victim should be brought into a warm room as soon as possible, given a warm drink, and either wrapped in warm blankets or placed in a tub of warm, not hot, water. Water is faster, for just as you lose more heat from your body in cold water, so your body absorbs heat more rapidly from warm water. Too much heat should be avoided: do not use a heat lamp or hot-water bottle; do not expose frostbitten areas to a hot stove; and do not rub or massage a frozen finger, toe or ear; but after the part is warmed, encourage the victim to exercise his fingers and toes.

The best approach to frostbite, however, is to prevent its occurrence in the first place. Dress warmly enough. Dress *dryly* enough. Exercise to keep warm, especially your toes and fingers. Do not drink alcoholic beverages or smoke during or immediately before severe exposure. And do not be one of those foolhardy heroes who have not sense enough to come in out of the cold.

WORKMEN'S COMP PAYROLL AUDITS

The *net* premium paid for Workmen's Compensation insurance is determined at the end of the policy year by an audit of your actual payroll. The premium you pay at the *beginning* of the policy year is an estimated premium which is based on an estimated payroll. A plus or minus adjustment is made in premium as a result of the payroll audit at the end of the policy year.

From payroll audits of some association members, it has been found that they are paying higher premiums than necessary for Workmen's Compensation insurance because of the manner in which they compute and record their payroll.

The premium for workmen's compensation is based on straight-time pay for all hours worked and should not include ½ extra pay for overtime hours worked.

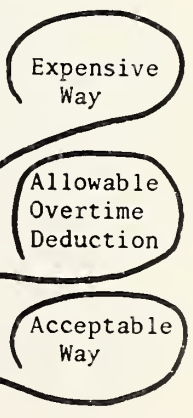
Many members are still using the expensive way (from the Workmen's Compensation premium point of view) to compute and record their payroll.

The proper breakdown of payroll may help you reduce premium costs. Example:

WARNING: Do not expect the Workmen's Compensation auditor to refigure your payroll for you, using the correct straight-time method. The Compensation Manual, Page R5, provides, in substance, that the entire remuneration shall be used as a basis for premium. The exception is that if the employer's books and records are kept in such a manner (by employee and class of work) that the amount of overtime pay is readily available in total and in summary for policy period, then the overtime pay (this is the amount earned in excess of the regular pay rate) may be excluded from the premium computation.

The Compensation rules further state that these totals must be available and presented to auditor at time of call. If not, the policyholder is not permitted any later or retroactive adjustment after audit is made.

Total Hrs	Straight Time			Overtime (Time & One-Half)			Gross Wages
	Hrs.	Rate	Amount	Hrs.	Rate	Amount	
54	40	$3.00	$120.00	14	$4.50	$63.00	$183.00
				Less: 1/3rd of overtime pay			(21.00)
				Payroll Applicable to Premium			$162.00



Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

Personalities

Joyce Sumner, Hutchinson, was re-elected Secretary of the American Society of Anesthesiologists at the recent annual meeting in New Orleans.

Vernon James, Wichita, has received the American Academy of Pediatrics annual award for journalism for 1977. The $1000 award was made in recognition of his weekly newspaper column on parenting.

Norman Pullman, Wichita, spoke on "Acute Phase Care of the Burn Patient" at a recent meeting of the Wichita chapter, American Association of Critical Care Nurses.

Whitney VinZant, Wichita, was guest speaker at a recent meeting of Sedgwick County Medical Assistants Society.

H. R. Draemel, Salina, was available to answer questions from the public in conjunction with exhibits and hearing tests during "Deaf Awareness Week."

Victor Hildyard II, Colby, presented a lecture and film series on burns at Colby Community College.

E. J. Chaney, Belleville, was elected to the National Board of the Institute of Family Practitioners during the recent national convention in Las Vegas.

Lecturers for a recent medical series at Colby Community College included **Asher Dahl, George Marshall** and **LaDonna Regier,** all of Colby.

Robert J. Dockhorn, Shawnee Mission, was recently named president-elect of the Joint Council of Allergy and Immunology.

Millard C. Spencer, Topeka, has been appointed clinical assistant professor in diagnostic radiology at KUMC.

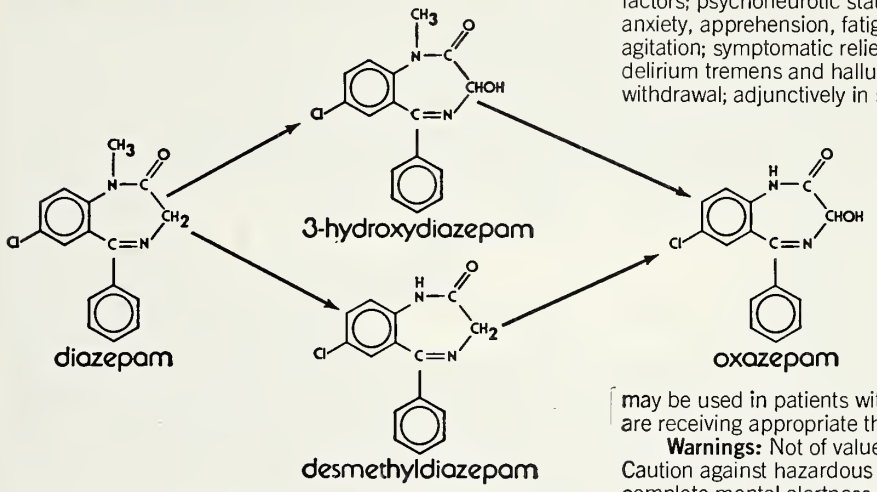
Paui B. Leffler, Pittsburg, has retired after 31 years of practice.

A medical library has been established at Ashland District Hospital in memory of the late **Ivan R. Burket,** Ashland.

John O. Yulich, Kansas City, was recently named clinical assistant professor in family practice at KUMC.

(Continued on page 67)

A pharmacokinetic character all its own



Valium (diazepam) is a benzodiazepine with a distinctive pharmacokinetic profile

The pharmacokinetic profile of Valium is one of the characteristics that sets it apart from other benzodiazepines. Consider, in particular, the metabolic pathway of Valium. The three major metabolites of Valium exhibit significant pharmacologic activity—and so, of course, does the parent substance—diazepam itself. All combine to produce the characteristic clinical response seen with Valium. The response you have come to know, to want and to trust.

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Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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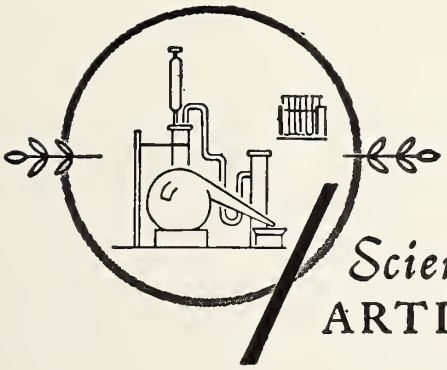
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Permanent Pacemaker

Incidence of Pacemaker Complications

SHELBY C. DICKERSON, M.D.; H. DANIEL LEWIS, JR., M.D. ***and***
ALFRED HEILBRUNN, M.D., ***Kansas City***

PATIENT populations with permanent implantable cardiac pacemakers have been previously reviewed and their complications outlined.[1–10] Unwanted surgery for problems related to pacemaker power packs has been mentioned in most of those series, and additional procedures for lead replacement or repositioning have also been outlined. The incidence of transvenous lead repositionings and the possible causes have been discussed.[7, 8, 11]

To assess the incidence of pacemaker complications and possible causes for transvenous lead repositionings in our hospital, we reviewed the records of 101 patients with permanent cardiac pacemakers seen at the Kansas City Veterans Administration Hospital through August 1976.

Records of patients with permanent cardiac pacemakers seen at the Kansas City Veterans Administration Hospital were reviewed to determine the incidence of complications with permanent pacing. The findings are analyzed and discussed.

Methods

We retrieved the hospital charts or operative records or both of all patients for whom these were available. Included were patients who received original permanent pacemaker implants or who were referred here for problems related to pacemakers that had been implanted elsewhere. The following information was obtained from each chart: (1) electrocardiographic or symptomatic indications for pacing; (2) dates of pacemaker implantations and replacement; (3) model numbers of the pacemakers and lead systems; and (4) length of follow-up. Pacemaker or lead system complications and causes of death in patients who died were also listed for each patient. Available chest x-rays were inspected for evidence of lead displacement or breakage, and cardiothoracic ratios (CTR) were determined as an index of heart size. In seeking reasons for repositionings, we compared CTR in repositioning and nonrepositioning groups; threshold currents at installation and at the time of repositioning were also recorded.

Results

The patients were all males, ranging in age from 43 to 85 years (mean 73 years). Most patients had fixed-rate pacemakers with transthoracic lead systems from 1963 to 1968; subsequently, most received demand pacemakers with transvenous leads.

Complete AV block was the most common electrocardiographic indication for permanent pacing *(Table I)*, occurring in 54 (58%) of the patients. Next most common were: sinus bradycardia (17%), slow ventricular rate with atrial fibrillation (9%), 2° AV block

Address reprint requests to Dr. Lewis, Veterans Administration Hospital, 4801 Linwood Blvd., Kansas City, MO 64128.

TABLE I

INDICATIONS FOR PACEMAKER IMPLANTATION

EKG Diagnoses	*No.*	*%*
Complete AV block	54	58
Sinus bradycardia	16	17
Slow ventricular rate in atrial fibrillation or flutter	8	9
2° AV block	5	5
SA arrest	5	5
Refractory ventricular tachycardia	3	3
Bifascicular block	1	1
Refractory supraventricular tachycardia	1	1
Symptoms	*No.*	*%*
Syncope or coma	39	46
Pre-syncope	9	11
Congestive heart failure	30	36
Angina	1	1

(5%), SA arrest (5%), and refractory ventricular tachycardia (3%). Episodes of syncope were the most frequent symptoms prompting permanent pacing (46%), with congestive heart failure (35%) and presyncope (11%) being relatively common. Not all of the patients' records mentioned a specific symptom as a reason for pacing.

Ninety-eight patients had follow-up for variable periods after pacemaker implantation *(Table II)*. Records for 59 patients were complete from the original pacemaker implant or referral until July 1, 1975. Many had not been seen after the review was begun, so records were considered complete only to that date. Prior to that time, 16 were lost to follow-up and 16 were known to have died after leaving the hospital. Five died later during the hospitalization for pacemaker implantation, and two had their pacemakers removed. Three other patients had original pacemakers implanted just after July 1, 1975, and had several months' follow-up. The patients' follow-up times were from one day to 12 years, with a mean of 56 months. We tabulated total patient-months of experience with permanent pacing to be used as an index in comparing different lead systems. The pacemaker experience of all patients totaled 3,043 patient-months.

A variety of causes of death were noted in the 16 patients who died after hospital discharge *(Table III)*. Many had other serious illnesses. Only Patient 3 had documented ventricular fibrillation. He had a fixed-rate pacemaker, and was listed as a "possible" pacemaker-related death. Patients 4 and 12 had demand pacemakers but died suddenly; no conclusion can be drawn as to whether pacing or pacing failure were causally related. In no case was a local complication involving a pacemaker mentioned as a cause of death. Five other patients *(Table IV)* died less than a month after pacemaker implantation; all died suddenly while still hospitalized. Two had ventricular arrhythmias documented terminally, but in none could pacemaker malfunction be proven to be a contributing problem.

Twenty-one patients experienced local complications involving the pacemaker power pack site *(Table V)*. Five had sterile effusions, and four had hematomas in the pacemaker pocket, all of which responded to drainage or evacuation. Both of these complications tended to occur early after pacemaker implantation. Scar breakdown, erosion, or trauma to the skin overlying the pacemaker affected six patients, all of whom required only local revision (with the local instillation of antibiotics in two instances). The two patients with sterile pleural effusions had recently undergone transthoracic lead implantation. Wound or pacemaker pocket infections (cultures positive for proteus or aerobacter bacteria in two cases) tended to occur late after pacemaker implantation. Only one of these required pacemaker

TABLE II

PATIENT-MONTHS

Category	*No.*	*Mean, Mos.*	*Range*	*Total Patient-Mos.*
Followed from original pacemaker to 7-1-75	59	56	1-144 mos.	2143
Lost to follow-up before 7-1-75	16	36	1-68 mos.	576
Died after leaving hospital	16	20	1.5-68 mos.	314
Died in hospital following pacemaker insertion	5	—	1-23 days	1
Pacemaker removed	2	—	1-9 mos	10
Total patient-months of pacemaker experience				3043

TABLE III
LATE DEATHS

Patient	*Age*	*Mos. After*	*Recorded Causes*
1	63	9	CHF
2	82	47	Sepsis
3	73	5	Ventricular fibrillation*
4	77	9	Sudden (100% LAD lesion at post)
5	83	40	Ca of colon, peritonitis
6	63	36	Shock after laprotomy
7	78	5	Diabetes, fever, sudden death
8	72	55	Aspiration pneumonia
9	65	60	COPD, bronchopneumonia
10	77	10	Lactic acidosis, CHF
11	77	1.5	Acute renal failure, resp. arrest
12	58	9	Sudden history of ventricular tachycardia, CHF
13	83	15	CHF
14	81	12	COPD, CHF, renal failure
15	69	43	CHF, acute renal failure, pneumonia
16	50	12	Aspiration of barium following abdominal surgery

* Recorded as "possibly" related to pacemaker.

TABLE IV
EARLY DEATHS

Patient	*Age*	*Days After Implant*	*Cause*
17	74	1	Sudden ventricular irritability recorded earlier
18	75	10	Sudden—unexplained
19	75	23	Sudden—unexplained
20	81	10	Sudden—cellulitis of left foot and known aortic stenosis
21	84	19	Refractory ventricular tachycardia and CHF

TABLE V
LOCAL COMPLICATIONS

Complication	*No. Patients*	*Time After Implant*	*Treatment*
Serous effusion in pacemaker pocket	5	1 day-2 weeks, and 8 months in one case	drainage
Hematoma in pacemaker pocket	4	12 hrs.-1 week	surgically evacuated
Scar breakdown or trauma to pocket	6	8 days-19 mos.	pocket revision (local antibiotics in 2)
Sterile pleural effusion	2	less than 1 week	none
Wound or pocket infection	3*	3 mos.-23 mos.	drainage and local antibiotics new pacemaker in one

* Two cultures showed proteus and aerobacter bacteria.

TABLE VI
PREMATURE PACEMAKER DYSFUNCTION

Patient	*Event*	*Time After Implant*	*Pacemaker*
1	Runaway pacemaker (160 BPM)	20 mos.	5870
2	Pacemaker rate increase (94 BPM)	18 mos.	Unknown Medtronic demand, implanted elsewhere
3	Fixed rate pacer on T wave with multiple PVC's	3 mos.	5860
4	Pacemaker sensing "P" waves	21 mos.	5841

TABLE VII
LEAD REPLACEMENTS

Lead	*Replacement*	*No.*	*Previous Repositioning*
Transthoracic	Transthoracic	0	———
Transthoracic	Transvenous	3	———
Transvenous	Transthoracic	4	4*
Transvenous	Transvenous	5	2

* One patient had replacement with transvenous lead previously.

replacement, and the other two infections resolved with the use of locally instilled antibiotics and surgical drainage, without systemic antibiotics.

Four patients had documented pacemaker dysfunction at 21 months or earlier *(Table VI)*. All of these pacemakers were older models which are no longer in use. Pacemaker rate increases would not be expected with newer pacemaker models.

Lead replacements or repairs were sources of additional unwanted surgery. Eleven patients had lead replacement *(Table VII)*. Three were converted from transthoracic to transvenous leads. Four were converted from transvenous or transthoracic leads after previous repositioning of transvenous leads in each. Of the five patients who had new transvenous leads installed, two of them had earlier required transvenous lead repositioning. The other three had myocardial perforation, broken insulation and a defective connection, respectively.

The 21 patients who required transvenous lead repositionings represented the largest group of patients with unwanted additional pacemaker related surgery *(Table VIII)*. Thirteen patients had "exit block," defined as the presence of a much higher threshold than at lead installation, without lead dislodgement visible by chest x-ray. In all of these, pacing resumed when the lead was repositioned. Eight other patients experienced displacement of a lead from its original position, as documented by chest x-ray, and normal pacing was obtained by lead repositioning. One patient underwent a total of three repositionings, and three others had two repositionings each. One of these ultimately received a replacement epicardial lead system.

Among the patients who had their original pacemakers installed at the Kansas City Veterans Administration Hospital, 75 had transvenous leads with the others receiving epicardial lead systems. Seventeen of the patients who had leads implanted here required lead repositioning, for an overall repositioning rate of

TABLE VIII
REASONS FOR TRANSVENOUS LEAD REPOSITIONING

	Model No.				
	5816	5818	5819	6904	OTHER
Exit block (total = 13)	3	3	3	1	3
Lead displacement seen by x-ray (total = 8)	—	3 & 2*	1	1	1

* Two were iatrogenic, one malpositioned in coronary sinus, another pulled accidentally during pacemaker replacement.

TABLE IX

THRESHOLD CURRENTS AT TIME OF PACEMAKER IMPLANTATION

	Patients With Later Repositioning (N = 10)	*Patients With No Repositioning (N = 14)*
Range (MA.)	0.5-1.5	0.1-1.8
Mean (MA.)	1.025*	1.007*
S.D.	± 0.121	± 0.123

* Difference insignificant at P > 0.1. (Student's T-Distribution.)

TABLE X

CARDIOTHORACIC RATIO FROM PA CHEST FILM

	With Lead Repositioning	*Without Repositioning*
No.	18	14
Range	0.42-0.68	0.42-0.69
Mean	0.53*	0.52*
S.D.	± .068	± .075

* Difference insignificant at P > 0.1. (Student's T-Distribution.)

23 per cent in our patients. Four other patients who underwent lead repositioning had their leads installed at other hospitals. Eight of the 17 patients experienced displacement of their transvenous leads visible by chest x-ray, for a repositioning rate of 11 per cent in this sub-group.

Threshold current measurements at original lead implantation were available for small groups of patients both with and without subsequent lead repositioning *(Table IX)*; there was no significant difference in thresholds at the time of lead implantation between these two groups. With no difference in the groups, and all thresholds below 2.0 ma, the implantation threshold had no predictive value for repositioning.

Similarly, cardiothoracic ratios from patients, both with and without lead repositioning, had a wide range of values and no significant difference in the mean for the two groups *(Table X)*. Patients both with and without lead repositionings were also found to be with and without enlarged hearts. These cardiothoracic ratios were measured from films nearest the time of a lead implantation or repositioning. It was found that the cardiothoracic ratio, as an index of over-all heart size, had no predictive value as to which patients will most likely need lead repositioning.

There were differences in the repositioning rate among the various leads in use *(Table XI)*. Most of the leads were Medtronic* and included models 5816 and 5818 with large tip electrodes; 5819 with a tapered tip but small tip electrode area; and 6904 with a large tip but small tip electrode area. All of the leads were bipolar. From the tabulated results, it is apparent that the older models had greater mean months of use per patient. Therefore, only the first ten months of use for any lead were considered for the purpose of comparison. Model 5819 tapered tip small electrode area lead had a higher rate of repositioning per patient month (0.076) during this period; while the 6904 lead with a small electrode area but larger tip had a lower rate (0.015). Intermediate values were observed for the older large tip electrodes. However, the number of patients with each lead was small, so no statistical significance can be inferred.

* Medtronic® leads, Medtronic, Inc., 3055 Old Highway Eight, Minneapolis, MN 55440.

TABLE XI

PATIENT-MONTHS OF EXPERIENCE WITH TRANSVENOUS LEADS

	Model No.			
	5816	*5818*	*5819*	*6904*
Tip size	4mm	3.2mm	2.2mm	3.2mm
Tip electrode area	85mm^2	53mm^2	11mm^2	15mm^2
No. patients	5	20	8	15
Mos. use, range	0.75-66.0	0.25-72.5	0.1-20.5	0.5-22.0
Mos. use, mean	31.6	26.8	9.8	9.2
Total patient-months	158	535	79	138
No. repositionings	3	10	6	2
Mean months before repositioning	23.0	13.5	2.5	1.0
Repositionings/patient-months in first 10 mos.	0.049	0.031	0.076	0.015

Discussion

Indications for permanent cardiac pacing in our group of patients were most frequently syncope or congestive heart failure, as in other reports.[2, 9] The most frequent electrocardiographic findings—complete AV block and sinus bradycardia—are also consistent with those in other series of patients receiving permanent pacemakers.[1, 8, 9]

Patient deaths were generally related to other diseases, but the five instances of sudden death during hospitalization raise the possibility of pacemaker-induced ventricular arrhythmias, even though they were not proven in the two cases monitored. Sudden death occurred in three patients who had received older fixed-rate pacemakers, including one who died after his discharge from the hospital.

Our patients had a low incidence of infections involving the pacemaker power pack.[12] Two of the three were managed with surgical drainage and local antibiotic irrigation; one required pacemaker replacement. Few patients had sterile effusions distending their pacemaker pockets, and most of these occurred soon after pacer implantation.

Repositioning of transvenous leads was the most frequent unwanted surgical intervention for our patients. Our rate of 23 per cent includes the patients whose transvenous leads were implanted here. This repositioning rate is high compared to other series, ranging from 1.5 per cent in one of Parsonnet's groups to 22 per cent in others.[10]

Both early and late repositionings were observed in our group *(Table XI)*; mean time of follow-up for transvenous leads was 18.5 months and mean time from lead implantation until repositioning was 10.5 months. The longer follow-up of many of our patients' transvenous leads may have added to our repositioning rate. It was noted that lead malposition problems tended to occur early and exit blocks requiring lead repositioning appeared later in most instances.

Neither implantation threshold nor heart size, as predictors of subsequent transvenous lead repositioning, had previously been analyzed systematically. Our small number of cases indicates that there is no relation between these two factors and a future need for lead repositioning.

We observed a decreased incidence of lead repositioning among patients who had 3.2 mm tip, small electrode area leads, as compared to the tapered tip small electrode or the large electrode leads. This may be related to high current density plus adequate lead stability.

Summary

We reviewed the records of patients with permanent cardiac pacemakers seen at the Kansas City Veterans Administration Hospital to determine the incidence of complications with permanent pacing. Most pacemaker related problems were treated conservatively, with power pack replacement needed only in the few instances of electronic malfunction. The largest group of complications were transvenous lead repositionings, and neither heart size nor pacing threshold at implantation predicted these.

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Hidden Meaning

Commonly Overlooked Allergic Disorders

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EVERY CLINICIAN, regardless of specialty, sees a large number of allergic patients. If his practice represents an average segment of the population, about 10 per cent of his patients experience allergy in a severe form and about 50 per cent in a minor form. Unfortunately, some types of major allergy and many types of minor allergy are easily overlooked. It is the purpose of this paper to draw attention to such neglected allergic diseases. The topics to be discussed are arranged in alphabetical order, making up a mini-encyclopedia.

Abdominal Pain. Recurrent abdominal pain of allergic origin is probably the most widely overlooked of all allergic complaints.[1] The pain, usually dull and accompanied by cramps, involves chiefly the upper-right quadrant, radiating to the flank. At times it is more generalized, but the lower abdomen is not usually affected.

Often the clinical picture mimics chronic cholecystitis. In these cases most of the pain, which may be little more than vague discomfort, extends from the right hypochondrium into the right side of the back. Frequently the patient experiences clay-colored stools, but with neither jaundice nor dark urine. Most of the patients we see have already had a work-up for organic disease of the gastrointestinal tract. If not, we insist that this be done before food allergy is considered.

Aspirin Allergy. Other than penicillin, aspirin is the drug most likely to cause an allergic reaction and, as with penicillin, the reaction tends to be severe.[2] Aspirin allergy should be suspected in all patients with asthma, urticaria, and angioedema (angioneurotic edema). It may also be the basis of almost any other allergic manifestation including headache, nasal congestion, and eczema. Some of the more common preparations containing aspirin are listed in *Table I.*

Breath Odor. The massive use of mouth washes in America is indicative of the prevalence of unpleasant breath odor. There are many causes, but among the most common is allergy. It may be a complication of allergic rhinitis, especially when polyps are present, but is more commonly a result of food allergy. Milk is the most common offender, but an occasional case is caused by egg, chocolate, or other food.

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Some of the commonly overlooked allergic disorders seen in every physician's practice, most of which can and should be handled by the patient's own physician, are presented in a "mini-encyclopedia" form.

Caffeine Intolerance. Whether caffeine causes true allergy is questionable, but it frequently causes such annoying side effects as irritability, insomnia, headache, tremor, and tachycardia. Some of the products that contain caffeine and their approximate content are shown in *Table II.*

TABLE I

Common sources of aspirin are:

Alka-Seltzer	Equagesic
Anacin	Excedrin
Anahist	Fiorinal
APC	Midol
Ascriptin	Pamprin
Aspergum	Quiet World
B-C	Sinaid
Bufferin	Sineoff
Cope	Triaminicin
Coricidin	Trigesic
Darvon Compound	Vanquish
Dristan	Zactrin
Empirin	

Patients who must avoid aspirin should be warned to look for the word *acetylsalicylic acid* on the labels of all over-the-counter drugs, and to check with nurses and pharmacists before taking prescription drugs. Aspirin is especially likely to appear in cold tablets, headache remedies, and other types of mixtures designed to relieve pain and the symptoms of respiratory infections.

TABLE II

COMMON SOURCES OF CAFFEINE

Product	*Amount of Product*	*Caffeine*
Coffee	1 cup (8 oz)	120-160 mg
Tea	1 cup (8 oz)	64 mg
Cola drinks	12 oz	36-48 mg
Chocolate	Candy bar	50-75 mg
Caffeine sodium benzoate	500 mg	250 mg
Cafergot	1 tablet	100 mg
Excedrin	1 tablet	65 mg
Fiorinal	1 tablet	40 mg
Empirin compound, Sinarest, Soma, Triaminic	1 tablet	30 mg

Canker Sores (Aphthous Stomatitis). Although canker sores are commonly caused by allergy to foods, this possibility has received little attention in the literature. The most common offenders in our experience are fresh tomato, orange (and other citrus fruits), apple and apple vinegar (commonly used in the processing of cucumber pickles), and chocolate. Another possible cause is toothpaste. Certain foods seem to have an inherent tendency to cause mouth soreness without ulceration. Common examples are avocado, sharp cheese, banana, pineapple, walnut, and pecan.

Colic (infantile). When an infant cries repeatedly from obvious abdominal pain, the possibility of milk allergy should be considered.[3,4] This possibility is strengthened if the infant exhibits other evidence of allergy, such as excessive sweating (especially about the head), nasal stuffiness, and perianal irritation. Some infants experience increased bronchial mucus with cough and wheezing indicative of asthma, a disorder of far more concern than colic or other feeding problems.

Soy formulas do not necessarily solve the problem, as soybean is itself a common allergen; consequently a meat base formula is usually the best substitute. Any other food the baby is given should also be suspect.

Colitis. The allergist must take strong exception to the traditional view that mucous colitis and spastic colitis are nothing more than idiopathic or psychosomatic diseases.[5,6] Food allergy should always be considered; the patient himself (or more commonly *her*self) often has a reasonably accurate idea of the responsible foods. Allergy should also be an early consideration in ulcerative colitis.

Constipation and Diarrhea. Much of what has been said about colitis applies also to these two complaints. Constipation should always suggest allergy to milk, especially in children, and in adults whose constipation dates from childhood. If these patients also have foul breath, they tend to attribute it to constipation, whereas milk is often the cause of both complaints.

Diarrhea, although less common than constipation as a manifestation of food allergy, is of greater concern to the patient and is much more annoying. Many patients are aware of offending foods but are not always careful in avoiding them. Some patients alternate between constipation and diarrhea.

Drug Allergy. If a patient who is taking a drug—either prescribed or over-the-counter—develops a new symptom, it may relate to: (1) the disease under treatment; (2) the drug being ingested; or (3) a cause unrelated to either. However, it is mandatory that allergy or other side effect be considered. The *PDR,* which every physician has, and *AMA Drug Evaluations,* which every physician should have, are excellent sources of information on all types of drug intolerance.

Drugs may cause any type of allergy, but the most common are those affecting the skin. *Table III* lists important skin manifestations of drug allergy and the common causes of each.

Enuresis. Although allergy seems unlikely, it sometimes is a cause of this distressing problem.[7] Milk is again the most common allergen, but other foods may sometimes be implicated. Inhalant allergy may also be a factor, and an added benefit to parents is the relief of enuresis sometimes realized through treatment of inhalant allergy.

Headache. Headache is much more common than asthma and urticaria in allergy practice. Inhalant and food allergy should always be considered as possible contributing factors in both simple headache and classic migraine.[8] The common inhalants are molds and house dust; the common food allergens are milk, chocolate, corn and corn products (especially corn syrup), onion and garlic, egg, legumes, and pork.

Middle Ear Disease. Allergy frequently plays an important part in recurrent infectious otitis media and in secretory (serous) otitis media.[9] The tendency of milk allergy to cause increased secretion of mucus is of the greatest importance here. It is our policy to withdraw milk from the diet for at least three months in all children with middle ear problems. Early attention to allergy may preclude the necessity for insertion of tubes into the middle ears.

Milk Allergy. The prevalence of milk allergy in both children and adults always impresses students of the University of Missouri at Kansas City and the Kansas City College of Osteopathic Medicine who elect to

TABLE III

IMPORTANT SKIN MANIFESTATIONS OF DRUG ALLERGY AND THEIR COMMON CAUSES

Acne: Bromides, chloral hydrate, corticosteroids, iodides, isoniazid, nalidixic acid (NeGram), oral contraceptives, quinine.

Eczema: Aspirin, barbiturates, methyldopa, phenothiazines, sulfonamides, warfarin.

Erythema Multiforme: Aspirin, barbiturates, bromides, chloral hydrate, chlorpropamide, codeine, estrogens, griseofulvin, insulin, oral contraceptives, penicillin, phenolphthalein, phenothiazines, phenylbutazone, sulfonamides, tetracyclines, tolbutamide.

Photosensitivity: Chlordiazepoxide (Librium), chlorpropamide, diphenhydramine (Benadryl), griseofulvin, nalidixic acid, phenothiazines, protryptiline (Vivactin), sulfonamides, tetracyclines (especially Declomycin), thiazides.

Rashes (exanthems): Aminophylline, aspirin, barbiturates, bromides, chlordiazepoxide, codeine, griseofulvin, hydantoins (including Dilantin), indomethacin (Indocin), insulin, iodides, isoniazid, meprobamate, oral contraceptives.

Urticaria: ACTH (corticotropin), aminophylline, aspirin, barbiturates, bromides, cephaloridine, chloral hydrate, chlorpropamide, clindamycin, codeine, digitalis, erythromycin, griseofulvin, heparin, hydantoins, imipramine (Elavil), indomethacin, insulin, iodides, isoniazid, meperidine (Demerol), meprobamate, methyldopa, morphine and other opiates, nitrofurantoin (Furadantin), oral contraceptives, penicillin, phenolphthalein, phenothiazines, phenylbutazone, propylthiouracil, sulfonamides, tetracyclines.

study clinical allergy with us. Milk allergy should be suspected in any patient who has unexplained headache, bowel disturbances, abdominal pain or distension, foul breath, recurrent middle ear infection, serous otitis media, excessive sweating, or almost any of the disorders previously mentioned. We routinely eliminate milk on a trial basis for at least three weeks to allow the patient (or his mother) to determine whether it must be avoided.

Nicotinism. T. S. Szasz, a psychiatrist, writes, "Psychiatrists . . . speak of 'alcoholism,' regard it as a mental illness, and offer cures for it, but they do not speak of 'nicotinism' and do not regard it as a disease."[10] Except for chronic obstructive lung disease, allergists do not see the baleful late effects of cigarette smoking, but they find that allergic patients who smoke are remarkably sensitive to their own smoke and to that of those around them. This has been shown to be true of non-allergic patients as well. What is the answer? We find that many smokers will give up the habit if they are properly approached, and when they do they are astonished at how much better they feel. Many patients begin smoking in junior high school and have forgotten the feeling of well-being of the non-smoker.

Odors. Every physician has patients with a severe intolerance of various odors. Occasionally this is a true allergic reaction; patients who are allergic to penicillin sometimes react in this way. More often it is an intolerance or idiosyncrasy. Common symptoms are headache, nasal congestion, wheezing, and occasionally vague irritability and depression. The most common offender is tobacco smoke, but also important are certain heavy perfumes, oil paint, paint thinner, traffic odors, and asphalt.

Poison Ivy. The patient himself usually recognizes this type of contact dermatitis if it occurs in the summer. It is often overlooked in the winter because it is not realized that although the plant has shed its leaves, the stems and roots remain very much alive all winter long likely affecting children who climb trees, woodcutters, hunters, and campers. A common source of involvement in winter is handling firewood. Exposure to the plant may be indirect, as the active ingredient may be present on clothing, shoes, dog hair, or garden tools.

Pruritis Ani. There are many causes of this distressing symptom. If other causes are ruled out, the patient should watch his diet to see if he can detect the responsible food. One of the most common and severe causes of pruritis ani is black pepper. Other common offenders are raw fruits, especially citrus fruits, tomato, apple, lettuce, and radish. If, through carelessness or by accident, the patient consumes a food known to cause distress, the anal area should be lavaged with plain water after every bowel movement. This procedure should be continued over a period of two or three days. Soap and vigorous drying with toilet tissue are to be avoided.

Ragweed Asthma. Although mild ragweed hay fever may sometimes be treated successfully with drugs, more severe cases should receive at least three series of hyposensitization, beginning each year in June. (There is considerable variability among allergists as to the time of beginning treatment; perennial treatment is still used to some extent.) The primary purpose of hyposensitization is to relieve, or at least modify, the nasal and conjunctival symptoms; but it also is of vital importance in the prevention of ragweed asthma, a type that may be violent.[11]

Tension-Fatigue Syndrome. Allergic diseases were long considered to be of psychogenic origin for reasons readily apparent to those who deal with allergic pa-

(Continued on page 61)

Dwarfism—An Overview

Recent Developments in Diagnostic and Genetic Concepts

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DWARFISM has been treated as a medical curiosity throughout history. However, in the past two decades there has been an awakening of interest in the study of growth problems which has facilitated a broadening of the general understanding of dwarfism. Technically dwarfism refers to individuals with disproportionate short stature, which is usually caused by a skeletal abnormality, as opposed to those with proportionate short stature, which usually results from a variety of other causes. Contrary to the long held belief that all dwarfism is caused by two diseases—achondroplasia (those with short limbs) or Morquio syndrome (those with short trunks)—it is now clear that there are well over 75 distinct disorders in which dwarfism occurs,[1–3] each with its own particular set of clinical and pathological features. These are not disorders of bone but of cartilage, and are known collectively as the chondrodystrophies. However, many have extraskeletal features as well. This article will provide a brief overview of this subject and acquaint the reader with some important aspects of dealing with dwarfism.

Disorders

Since a detailed discussion of each disorder and its manifestations is far beyond the scope of this paper, *Table I* is given as a reference. This list is limited to a few of the more common chondrodystrophies and to a few salient features of each. There may be considerable clinical overlap among similar disorders as well as variability within a single disorder, both of which must be considered when trying to differentiate specific entities.

Nomenclature and Classification

The rapid progress made in delineating specific disorders has resulted in much confusion regarding nomenclature and classification. For example, naming has been based on a variety of schemes including: (1) the portion of the bones affected radiologically, *i.e.,* epiphyseal dysplasias, metaphyseal dysplasias; (2) a Greek term describing the appearance of the bone, such as diastrophic (twisted) dwarfism, or the course of the disease, as thanatophoric (death seeking) dwarfism; (3) an historical term, such as achondroplasia; or (4) an eponym, *e.g.,* Kniest syndrome or Morquio syndrome. Similarly, many systems have been employed for classification. The currently used scheme relies primarily on the predominant site of radiologic involvement (epiphyses, metaphyses, diaphyses, vertebrae). However, in recent years, morphologic, histochemical, and ultrastructural abnormalities of the endochondral growth plate have been identified for many of these disorders.[3–7] From this data, it is now possible to tentatively classify them according to the stage of endochondral ossification which is disturbed.[8] Thus, disorders will someday be named and classified on the basis of their pathophysiology which should bring considerable order to this confusing area.

Causes and treatment of short stature resulting from skeletal abnormalities are presented and a standardization of terminology is advocated.

Causes

It appears that a large number, perhaps most, of the chondrodystrophies have a genetic basis. Many represent single gene defects and are inherited in a classical Mendelian fashion (*e.g.,* achondroplasia, autosomal dominant; metatropic dwarfism, autosomal recessive; SED-tarda, x-linked). Others are suspected of being due to single gene defects, but their inheritance cannot be proven. For example, many of the forms of dwarfism which are lethal in the neonatal period, such as thanatophoric dwarfism, occur sporadically and may well represent new autosomal dominant mutations. Similarly, some of the very rare forms of dwarfism may represent autosomal recessive traits in which too few affected individuals have been recognized to establish an inheritance pattern.

Pathogenesis

Nearly all forms of dwarfism result from defects in endochondral ossification. This process is responsible for the embryologic development and subsequent linear

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TABLE I

PARTIAL LIST OF CHONDRODYSTROPHIES

Disorder	*Inheritance**	*(Age) Diagnosis*	*Major Clinical Features*	*Complications*	*Specific Histopathology at Growth Plate*
Achondroplasia	AD	Birth	Short limbs, bowed legs, stubby fingers, bulging forehead, sunken face	Hydrocephalus (infant) lumbar spinal cord compression (adult)	No
Hypochondroplasia	AD	Childhood	Similar to achondroplasia but milder (may approach normality)	Usually none	No
Multiple epiphyseal dysplasia†	AD	Childhood-young adulthood	Short limbs (mild)	Precocious osteoarthritis‡	No
Spondylo-epiphyseal dysplasia—congenita	AD	Birth	Short trunk, barrel chest, myopia, cleft palate	Subluxation of cervical spine due to odontoid hypoplasia, retinal detachment, precocious osteoarthritis‡	Yes
Spondylo-epiphyseal dysplasia—tarda	XL	Childhood	Short trunk (mild)	Precocious osteoarthritis‡	No
Spondylo-metaphyseal dysplasia	AD	Infancy	Short trunk, barrel chest, bowed legs	Usually none	No
Metaphyseal chondrodysplasia—Schmid type	AD	Childhood	Short limbs, bowed legs	Usually none	Yes
Metaphyseal chondrodysplasia—McKusick type (cartilage-hair-hypoplasia)	AR	Late infancy	Severe shortening of trunk and limbs, sparse blond hair, light complexion, immune deficiency	Squamous cell carcinoma of skin, complications of viral infections	Yes
Metatropic dwarfism	AR	Birth	Short limbs, severe progressive kyphoscoliosis, large joints, joint limitation	Usually none	No
Kniest syndrome	AD	Birth	Short limbs, kyphoscoliosis, large joints, flexion contractures, cleft palate, myopia	Retinal detachment, precocious osteoarthritis‡ with contractures	Yes
Pseudoachondroplasia§	AD	Early childhood	Short trunk and limbs, ligamentous laxity, bowing of long bones (forearm)	Precocious osteoarthritis,‡ flexion contractures at knees	Yes
Diastrophic dwarfism	AR	Usually birth	Short limbs, kyphoscoliosis, joint contractures, club foot, hitchhiker thumb, cauliflower ear	Precocious osteoarthritis‡ with contractures	Yes
Thanatophoric dwarfism	Sporadic	Birth	Short limbs, small thorax	Lethal in perinatal period	Yes
Achondrogenesis¶	AR	Birth	Short limbs, small trunk	Lethal in perinatal period	Yes

* AD—autosomal dominant, AR—autosomal recessive, XL—X-linked inheritance.
† Two types: Fairbanks—involves most epiphyses; and Ribbing—milder, often involves only capital femoral epiphyses.
‡ Precocious arthritis involves weight bearing joints, particularly the hips.
§ Probably several types.
¶ Two types: Parenti-Fraccaro and Langer-Saldino.

growth of most bones of the skeleton, and is thus the major process contributing to increase in height. Briefly, the process consists of: (1) an orderly proliferation of chondrocytes to form linear columns; (2) maturation of these cells with synthesis and secretion of large amounts of cartilage matrix; (3) degeneration of these cells concomitant with the calcification of adjacent matrix septa (provisional calcification); (4) cell death with rupture of the metaphyseal capillaries into the chondrocyte lacunae, and deposition of bone matrix upon the calcified cartilage spicules; and (5) complete resorption of calcified cartilage matrix and its replacement by bone matrix within the metaphysis.[8] Because of the sequential nature of this process, virtually any defect will lead ultimately to short stature due to reduced linear bone growth. In addition, depending on the site of the defect, a variety of other skeletal deformities occur as well.

When the basic defect is limited to the chondrocyte, the skeletal dysplasia is the sole manifestation of the disorder. However, in many cases, the defect is a generalized abnormality of the connective tissue which is shared by the cartilage. In these cases the skeletal dysplasia is only a single component of a generalized disorder, occurring with a variety of associated problems such as myopia, cleft palate, and club foot in SED congenita. The majority of the chondrodystrophies probably fall into this latter category.

Evaluation

It must first be determined that the individual is truly short. When growth charts are carefully examined and parental heights considered, it is often found that the patient is growing normally for his genetic potential, although not necessarily as much as the patient or his parents would like. Having established growth failure, proportionality must be assessed since proportionate short stature is usually due to nonskeletal abnormalities such as nutritional deficiency, endocrine imbalance or chronic disease, and the evaluation will be different for each. If skeletal disproportion (true dwarfism) is determined, a skeletal abnormality is likely, and complete skeletal radiographs are required. Radiographs taken prior to epiphyseal closure (puberty) are generally more helpful than those taken during adulthood. A comprehensive family history should be obtained and family members examined when possible; this process sometimes results in the identification of mildly affected family members, and this may help to establish the diagnosis. The combination of clinical, radiographic, and genetic information may be sufficient for making a diagnosis. In addition, it is now possible to establish or confirm the diagnosis for many disorders on the basis of specific histopathologic changes in the endochondral growth plate. The growth plate biopsy can be obtained at the time of therapeutic surgery, or alternatively an iliac crest biopsy can be obtained under local anesthesia. Also, in cases of lethal neonatal dwarfism, a diagnosis can often be made from autopsy growth plate material. As in other areas of medicine, it is likely that obtaining a tissue diagnosis will be an integral part of an evaluation of dwarfism in the future, particularly in rare disorders in which a definite diagnosis cannot be made otherwise.

Treatment

Unfortunately, there is no treatment to stimulate growth in any of the forms of dwarfism. Therapy is directed toward possible prevention or early treatment of skeletal and extraskeletal problems. Few dwarfs totally escape orthopedic problems, the most common being precocious arthritis of weight bearing joints, especially the hips. Prior to total hip replacement capability, many of these individuals were incapacitated as young adults; however, this procedure now often restores relatively normal activity. Abnormalities of the vertebrae may require surgical intervention such as correction of scoliosis in diastrophic dwarfism, laminectomy for lumbar spinal stenosis in achondroplasia, or cervical fusion to stabilize the cervical spine in SED congenita. Intervention prior to the development of such complications as spinal cord compression is obviously desirable. Since each disorder has its own particular orthopedic problems, it is of prime importance to establish the correct diagnosis so that specific problems can be anticipated. Likewise, complications of extraskeletal manifestations can often be prevented. For example, individuals with SED congenita and Kniest syndrome should have frequent eye examinations, because of their predisposition to retinal detachment. Treatment of early retinal tears may prevent the occurrence of total blindness which accompanies complete retinal detachment.

In addition to anticipation of problems, it is important to establish the correct diagnosis for two other reasons: (1) the physician can offer the patient or parents an accurate prognosis with regard to life expectancy, mentality, and anticipated height; and (2) proper genetic counseling can be provided. For example, consider young parents who want to expand their family but have just had a child with lethal neonatal dwarfism. If the diagnosis is thanatophoric dwarfism, then the risk for recurrence is negligible; on the other hand, if the child had achondrogenesis, an autosomal recessive disorder, the recurrence risk is 25 per cent for each subsequent pregnancy, a risk which many parents might

choose not to take. Thus, for a variety of reasons, it is extremely important to establish the correct diagnosis as early as possible.

KUMC Program

A short stature center dealing particularly with dwarfism has now been established at the University of Kansas Medical Center. The major function of the center is to provide care in terms of diagnosis, treatment, and genetic counseling to all individuals with dwarfism throughout the state. Until recently, there has been no facility dealing specifically with this problem in the state. In addition, investigations into many different aspects of dwarfism are ongoing, and clinical and radiologic data are being compiled. Since these disorders are individually rare, such data is very useful in further characterization, such as the establishment of growth curves. Growth plate specimens from patients with a variety of disorders are being studied by histochemical and immunochemical methods. This newly developed approach permits abnormalities in the distribution and content of the major components within the growth plate to be fully characterized. Such studies will very likely provide valuable clues concerning the pathogenetic mechanisms which are responsible for these disorders.

Summary

Dwarfism results when endochondral ossification is disturbed. There are over 75 distinct disorders, most of which are genetic, in which this occurs. Although there is no treatment to stimulate growth, many of the problems which in the past have been associated with dwarfism can be effectively treated and, in some cases prevented, by early diagnosis and treatment. Facilities to deal with all aspects of dwarfism (diagnosis, treatment, genetic counseling) have been established at KUMC.

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Hidden Meaning

(Continued from page 57)

tients. Persons afflicted with allergies often exhibit many symptoms that suggest neurosis, such as irritability, excitability, overactivity, restlessness, achiness, torpor, and fatigue. However, these are ways in which allergy commonly affects behavior, and treatment usually relieves them to a remarkable extent. Many a child receiving methylphenidate (Ritalin) or other behavior modifying drug is actually a victim of the allergic tension-fatigue syndrome and should be treated accordingly.[12] Far too often allergic adults report that their constitutional complaints have been written off as manifestations of a neurosis.[13]

Vulvovaginal Irritation. Allergic involvement of the sensitive and vulnerable female perineum may cause erythema, pruritus, increased vaginal secretion, burning on urination, and recurrent cystitis.[14] The offending allergens are much the same as those that cause pruritus ani.

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Retarded Children

An Update for Physicians on Program Alternatives

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CONVEYING the initial diagnosis of retardation and making suggestions for developmental programming represent a demanding and potentially frustrating experience for the clinician. In over 90 per cent of the cases, the physician is the first professional to notify the parents of their child's diagnosis.[1] This contact is the first opportunity for a professional to provide the family with advice and referral; however, frequently no referral is made. In a study by the Connecticut Department of Mental Retardation, only 24 per cent of children diagnosed as retarded received any referral.[2] In the same study, it was noted that while the average age of the child when retardation was suspected was 6.2 months, the average age of the child when the regional early intervention center was first contacted was 23.8 months. This 17.6-month delay between diagnosis and intervention is an alarming fact, especially considering our present knowledge of the significance of early intervention with infants who are high risk for retardation.[3, 4] It is possible that the medical profession has been unduly influenced by the tenets of Arnold Gesell, whose unsubstantiated theories regarding the futility of attempting to teach children developmental skills before they are maturationally ready may explain this unfortunate statistic. Rather than a wait-and-see approach, families of special children need information concerning accessible mental retardation clinics, agencies and parent groups, and this information should be given and the agencies contacted as early as possible.[1, 5] For a more detailed description of how physicians may interpret their findings to parents, see Solomons' work on parent counseling.[6] With physicians' increased knowledge of the efficacy of community alternatives to institutionalization which are now available, perhaps more effective information and referral delivery for retarded children and their families will result.

Early Childhood Research Group

One available program is the Early Childhood Research Group (ECRG) located in the Mental Retardation Research Center at the University of Kansas Medical Center. The ECRG was established in 1975 to serve three purposes: service, training, and research. The program provides treatment services for multihandicapped children aged six months to six years. Participating children have ranged from profoundly to mildly retarded with such medical diagnoses as cerebral palsy, psychomotor and growth retardation, Down's syndrome, child abuse, tuberous sclerosis, meningitis, and various seizure disorders. Many of the children were referred by community agencies which serve developmentally delayed children but who were unable to work with a particular child usually because of uncontrollable behavior such as tantrums, self-injury, or unresponsiveness. Physicians and their students in pediatrics, neurology, physical medicine, orthopedics, or psychiatry are consulted for assistance in management of the child's total health care.

Methods of developmental programming for retarded children are discussed and the importance of early referral is emphasized.

The second purpose of the ECRG is to train clinicians from a variety of disciplines to set up and carry out individualized educational program plans (IEPs) such as those described in Public Law 94-142.[7] This law requires that an individualized program plan be developed for every handicapped child in the public schools of every state including Kansas by 1979. Staff from the departments of nursing, psychology, occupational therapy, and speech pathology have participated in the training of students from these and other disciplines. Students learn to identify the child's current skill repertoire with regard to fine and gross motor, language, social, and self-help skills. Each skill area is subdivided into smaller specific behavior areas. These behaviors are then analyzed, defined, and broken down into small trainable units. (For a task analysis of walking, see Haavik and Altman.[8]) Students learn precise data collection techniques which permit data-based decisions about the adequacy of the child's program, ef-

fectiveness, correctness of the task analysis, and an accurate statement of the child's current level of functioning. The precise and objective data collection techniques enable different clinicians to conduct a specific child's treatment on separate days. Frequent recording of reliability (i.e., interobserver agreement) between clinicians provides an empirical check on the accuracy of data collection.

Required weekly group meetings are used to facilitate consistency in program planning or modification of each child's program in areas of task choice, sequencing of skills, motivational aspects, and behavior management problems. The group meeting is also used as an informal seminar where principles of developmental programming and behavior management are taught via discussion of case problems. Occasionally, particular topics chosen by students are also presented to the group by a faculty member or a guest speaker. The students may choose to focus on a particular training sequence, and videotapes are made of that sequence at least twice during a semester. The videotapes are then presented during the group meeting for feedback from faculty and other students.

Approximately every six weeks the clinicians summarize their data for the medical record, discuss the child's progress with parents, and help the parents decide whether the child should continue in ECRG or be transferred to another community program. These parent interviews are videotaped at least once each semester and played back to the student group to facilitate the teaching of clear and concise communication skills when interpreting to parents a retarded child's progress and potential capabilities. Home visits and parent training programs are also carried out by the student clinicians when indicated.

The ECRG staff design and carry out research projects which help delineate efficient and effective techniques for working with developmentally delayed children. Research to investigate the effects of diet on seizures and hyperactivity, the potential role of parents as clinicians to develop or maintain newly acquired skills in their own children, and methods to facilitate transfer of speech training from the therapy session to the natural environment is in progress. Past projects have included studies of techniques to facilitate walking and decrease disruptive and self-injurious behavior in handicapped children. Occasionally, interested medical students have assisted in designing and implementing research projects.

Summary

Physicians, as primary care providers, are key persons in the early diagnosis and referral of children who are at risk for mental retardation. Previously, costly delays occurred before children were referred for educational treatment. Current data supporting the effectiveness of early intervention provide physicians with an empirical basis for recommending immediate referral to a program such as the ECRG. For specific information regarding local programs, the physician may contact the special services division of the school system, the nearest office of Social and Rehabilitation Services (SRS), or consult the Resource Information Directory of Services to Handicapped Children in Kansas.[9]

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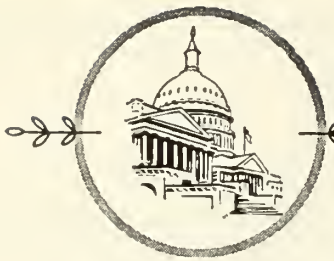
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Socio- ECONOMICS

Systematic Approach to Delegation

Ed Note: This is the fourth in the series of articles prepared by the AMA Department of Practice Management, Division of Medical Practice. For other articles on this topic, see the following issues: November, 1977, December, 1977, and January, 1978.

The subject of practice management has become more critical in recent years. To assist the physician in a smoother operation of his office, the Department has produced workshops for medical assistants dealing with such aspects as collections, public relations-telephone techniques and office management, as well as practice management workshops for the new physicians. The Kansas Medical Society Executive Office will present such workshops upon demand by members.

EVERYBODY talks about delegation—physicians who delegate the most seem to be the most productive—but how do you delegate in a medical practice? The theory is ideal; the doctor should spend as much time as possible doing only the things he is particularly equipped to do and then delegate the procedures that others can do as competently as he might do them. If they are delegated to staff at a lower income level than the doctor, it is economically advantageous, and if the doctor then uses the time to provide more "hands-on care" it will improve the quality of his patient care.

The question of "how to delegate" should be preceded by "why," "when," "what" and "to whom" should you delegate. The major purpose for delegating is to save time for the physician. If you delegate a task that takes only eight or nine minutes and is done only once a month, you have not accomplished very much. The time required to *properly* delegate such a task could be more than the time saved. In considering the delegation of any procedure the first question to ask is, "How much time will this save?"

It is equally important to consider "when" to delegate. The negative approach to this question, when *not* to delegate, is the most obvious. Do not wait until an epidemic or crisis situation—when the reception office is overflowing, all of the examining rooms are filled, and you have promised to take your spouse out to dinner to celebrate your anniversary and will not be able to leave the office before 8 PM—and then start delegating to any staff member available. The time to delegate is when you have plenty of time to train the staff person to competently perform the task you want done.

What tasks should you delegate? This is entirely up to you. Every physician has his own art form. A task that you might be willing to delegate, another physician might not be comfortable in delegating, and vice versa. The procedures that you delegate will depend on the way you practice medicine, the type of staff you have, the number of examining rooms and your office layout, and your particular patient load.

There are no instructions to tell you what specific tasks to delegate but there is a procedure you can follow that will help you select the tasks that can be delegated to your advantage, in your practice. Make a list of all of the tasks you do. If you identify each separate task, not just overall procedures, the list will probably be over 100 different items. Then carefully examine each item in the list and ask, "Does this task have to be performed by a physician?" Could it be delegated to a nurse or a medical assistant? If I delegated the task, would it save me time?"

You will probably find a number of tasks that could be delegated which you are doing because you did them when you started your practice; they are easy and comfortable to do and you developed a routine in which they readily fit. No one would suggest taking these tasks away from you if there is nothing to be gained. But if your practice situation has changed, if you now have more patients, if you are not spending as much time with each patient as you would like, or if you have added more examining rooms or more medical assistant help, then you might want to consider delegating the particular task. Do this with every item on the list and mark it accordingly.

Do not throw the list away. Save it and review it a year later. At that time, your practice situation may again be different and alter some of your decisions. Medical practice seldom remains static.

When you decide to delegate a task, to whom will you assign it? You will, of course, want to know that the staff person has the background experience or training, or the ability to learn to do the task competently.

After all, you are responsible for the actions of your employees. But a factor you do not want to overlook is the willingness of the person to learn to do this task. Most of the time you will find your employees are willing and eager to take on new jobs for the self-satisfaction that comes from increasing one's skills. This is one of the few things you can offer in "upward mobility." However, you may find an employee who would not be interested or comfortable in doing the particular task. You definitely would not want to assign the task to such a person, so the first thing you do is to ask the employee, "Would you be willing to learn to do this? Would you be comfortable in doing it?"

Assuming the person you have selected is most willing to do the task, we now come to the question of how do you delegate it. Delegation is more than just telling. To do it properly you need to follow one of the numerous job training textbook formulas that emphasize "show and tell," evaluation, and authorization limits. You start by writing a step-by-step, detailed description of the task. An easy way to do this is to dictate it and let the person who is going to do the task transcribe the dictation. After this has been done, your employee can become familiar with the step-by-step instructions from the written transcription. When you demonstrate, describe why and how you do each step; and in turn, have your employee do the task for you, with a similar step-by-step description. If it is a complicated procedure, establish a system for periodic evaluation so that you can be sure the task is being performed as well as you would do it. And finally, set some limits beyond which the person should not go in performing the task, but should report back to you. (For example, we know of a situation where a medical assistant having difficulty in finding the vein to draw blood jabbed the needle into the patient's arm 12 times.)

There is a management technique that encourages employees to use their own initiative, and we subscribe to that in the business office operation. Suggestions from the staff can be most valuable in developing efficient office procedures. However, in delegating a task that has been performed by the physician to an employee who has not done the procedure, it is essential for the physician to follow a step-by-step training technique. When he does this so that he is assured the task will be performed competently, he will save time for himself that can be used in providing patient care.

UNIVERSITY OF KANSAS COLLEGE OF HEALTH SCIENCES
DIVISION OF CONTINUING EDUCATION

Symposia:
March 13, 14 and 15, 1978
PEDIATRICS
Guest Faculty:
ROBERT B. JOHNSTON, M.D., Johns Hopkins University School of Medicine and John F. Kennedy Institute for Handicapped Children, Baltimore, Maryland.
FRANK J. MENOLASCINO, M.D., University of Nebraska College of Medicine, Omaha.
AUBREY MILUNSKY, M.B.CH., Harvard Medical School and Eunice Kennedy Shriver Center, Boston, Massachusetts.
CHARLES M. POSER, M.D., College of Medicine, University of Vermont, Burlington.
Subjects to be discussed will include: PRACTICAL APPROACH TO DIAGNOSIS OF MENTAL RETARDATION; CURRENT STATUS OF PRENATAL DIAGNOSIS; UNDERSTANDING MOTOR DISABILITIES; CHILDHOOD AUTISM—WHAT IS IT?; EPILEPSY—SOCIAL AND EDUCATIONAL IMPLICATIONS; DEVELOPING COMMUNITY RESOURCES FOR DEVELOPMENTAL DISABILITIES; FAMILY INTERVENTION IN SCHOOL PROBLEMS.
Accreditation:
American Medical Association: 20 hours of Category I Credit.
American Academy of Family Physicians: 20 hours of Prescribed Credit.
Registration Fee: $120.00

March 16 and 17, 1978
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Guest Faculty:
CHARLENE ANDERSON, R.N., Medical Representative, Traveler's Insurance, Overland Park, Kansas.
WILLIAM T. BAKER, Division of Vocational Rehabilitation, Topeka, Kansas.
JOHN W. GOLDSCHMIDT, M.D., Northwestern University Medical School and Rehabilitation Institute of Chicago, Illinois.
MARTHA MYERS, Panhandle Eastern Pipeline Company, Kansas City, Missouri.
P. K. WORLEY, M.A., Executive Vice President, American Cancer Society, Kansas Division, Inc., Topeka, Kansas.
Subjects to be discussed will include: WHAT IS CANCER? PATHOLOGY, DIAGNOSIS, COMPONENTS OF PROGNOSIS; GYNECOLOGIC CANCER; GASTROINTESTINAL CANCER; CANCER PROGRAMS IN KANSAS; COUNSELING PATIENTS AND FAMILIES WITH CANCER; LIFE FOLLOWIG MASTECTOMY; REHABILITATION NEEDS OF CANCER PATIENT; COUNSELOR AWARENESS OF CANCER.
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For program announcement and information, write: DIVISION OF CONTINUING EDUCATION, University of Kansas College of Health Sciences, Kansas City, Kansas 66103.

Physician Assistant

Breaking Ground in Topeka

ERIC SCHUMAN, P.A., ***Topeka***

The education of Physician Assistants and their role in contemporary medical practice is described by a PA with the Family Practice Group in Topeka.

"FROM THIS DAY on for the rest of your working life, for 24 hours each day, you are ambassadors of your profession." Thus was I greeted after being admitted to the Long Island University/Brooklyn-Cumberland Medical Center Physician Assistant Program in New York City, and these words were prophetic.

Prior to my arrival in Topeka in March 1976, no primary care Physician Assistant (PA) had been employed here. Robert E. Jacoby, M.D., a native Topekan and a graduate of KU Family Practice Residency, was forming a group of family practitioners, physician assistants and nurse clinicians, and hired me initially for a three-month period. Through a subsequent six-month evolvement, my role in the practice became defined as we learned by trial and consideration how a physician and a PA work together.

I am a primary care practitioner working basically in the office with an average of 14 patients each day. Their appointments with me are scheduled in advance, and my title and role are reinforced by our receptionists, nurses, and in our *Patient Information Booklet.* Patients are not screened by chief complaint or history, but they are given the option of seeing a physician, although this may mean a delay in their appointment. Charges for my services are identical to those of the physician since I am legally an extender of his practice.

PAs are trained in programs accredited by the AMA Council on Medical Education and standardized to include minimum curriculum requirements, such as basic college courses in biology, chemistry, physics, anatomy, and physiology. A semester is spent studying each of the human organ systems (gastroenterology, cardiology, urology, ophthalmology, endocrinology, pediatrics, obstetrics and gynecology) by reviewing each system's normal anatomy and physiology, common pathology and appropriate treatment, including relevant pharmacology. Clinical rotations are required in internal medicine, surgery, pediatrics, obstetrics/gynecology, and electives. The student then sits for a two-day examination administered by an arm of the National Board of Medical Examiners. It includes 1000 written questions (multiple choice and patient management problems) and one day of clinical skills in which the student performs a physical examination on three patients, each proctored by a physician preceptor. Included is examination of the abdomen, chest and eyes, and a neurological evaluation. PAs complete their training programs in two to four years, depending on program design and prior college experience. Many programs, such as the one at Long Island University, require three years of direct patient care experience prior to admission. There are two essential skill requirements an adequately trained PA should possess on graduation: (1) he should be able to secure an accurate history and perform a thorough physical examination; and (2) he should be able to recognize his own limitations.

Most primary care PAs are under indirect supervision while diagnosing and treating patients' illness; but the PA must be able to immediately recognize and bring to his physician-employer's attention any historical or physical finding, laboratory test, or subjective impression that indicates physician intervention. That point will be determined for each PA by varying factors, such as experience and training.

An analysis of our practice, performed by the University of Wisconsin Medical School (results available by request from the author), indicates that I make diagnostic and therapy decisions independently 75 per cent of the time—*i.e.,* without consultation or a physician's presence in the examining room. The percentage is higher in the office and considerably lower with hospitalized patients. Dr. Jacoby and I have a clear understanding of which findings require consultation: (1) the decision to hospitalize; (2) the use of a drug he would not normally prescribe in the same situation; and (3) referral to a specialist. My supervision is further assured by having a physician read and countersign all my charts and by our periodic intervals spent together

with patients.

A small percentage of my time has been spent working with our health educator designing programs for patient education in the office. In addition to the usual array of pamphlets, we have a videotape unit which we use to educate new mothers on well child care, and patients with chronic diseases such as obesity and diabetes. We produced our own videotape as part of a patient education prescription used routinely with hypertensives.

Numerous studies are available confirming that PAs are cost-effective and enjoy high patient acceptance. Approximately 50 AMA approved programs are graduating 2,500 PAs each year. Estimates in Kansas approach 250 practicing PAs, although only 68 are currently registered with the Board of Healing Arts. Most are working in primary care, although some are employed by specialists. Our roles are diverse; some work primarily with hospitalized patients and others never do.

The PA program was born in this country when Duke University graduated a class of four in 1967. The initial program addressed the question of how to utilize medical skills of ex-military corpsmen in civilian life. In 1970, the AMA encouraged the PA movement in the United States to direct qualified personnel into medically underserved areas, especially the urban ghettoes and rural communities.

Unfortunately, most PAs have not located in these settings. Federal and state governments have not employed PAs to work in areas of highest need, and the locations of physicians employing PAs have often reflected the general problem of medical maldistribution.

The issue of third party reimbursement for services rendered by PAs has also been a problem. Medicare regulations are vague and confusing, at best. Hospital services are almost never covered by third parties, but most office care is reimbursed fairly.

Malpractice has not been a problem for PAs, who can be covered by inexpensive riders to the employing physicians' policies, or independently through the American Academy of Physician Assistants. No judgment has ever been levied against a graduate of an AMA approved PA program in a malpractice suit. An article in one medical journal called the employment of PAs additional malpractice insurance for doctors, since PAs can often spend more time listening to patients than physicians can.

Our Academy requires 50 hours of CME each year. Without it we are required to sit for the Boards every two years. Even with required CME, we must take the examinations every six years.

PA salaries vary according to experience, with starting wages usually in the $12,000-$16,000 range.

My own experience with the medical community in Topeka has been overwhelmingly positive. Nurses in the hospitals have been kind and helpful. I enjoy a good relationship with Topeka's large number of extended-role nurses, and I meet regularly with them. Several specialists with whom we routinely consult meet with me in the hospitals and at grand rounds.

The need for mid-level practitioners in Kansas is great. Most of us find a practice setting with high physician accessibility ideal, but we recognize this is not always possible. Satellite health facilities staffed by PAs or nurse clinicians with remote supervision have been successful in other states and would likely succeed here as well. Our chief concern is the delivery of the highest quality medical care to as many Kansans as possible, now and in the future.

Personalities

(Continued from page 46)

Joseph H. Depoe has closed his offices in Douglass and Winfield to enter the Air Force.

Thomas Rosenberg, Wichita, recently spoke on child abuse at a meeting of the Modernettes of Whitewater.

K. J. Bierlein, Pittsburg, has phased out his 44-year practice in southeast Kansas.

The Kansas Chapter of the National Committee for Prevention of Child Abuse has named **Arthur Cherry,** Topeka, to its Board of Directors.

The Kansas Rehabilitation Association has honored **Neal M. Jenkins,** Salina, with a Health Services Award for his role in establishing and serving its vocational rehabilitation center at the former Schilling Air Force base hospital.

Daniel L. Azarnoff, Kansas City, was named to the Institute of Medicine, National Academy of Sciences.

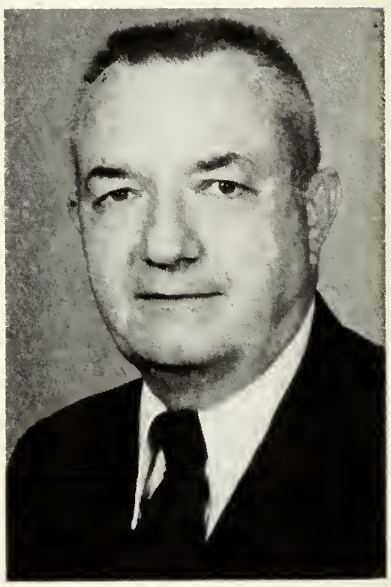
George Belcher, Columbus, recently addressed members and guests of TOPS in Columbus.

Don Tillotson, Ulysses, was the featured speaker at a recent meeting of the Grant County Division of the Kansas Chapter, Arthritis Foundation.

Jimmie Gleason, Topeka, was a panelist at a recent forum on patient-doctor relationships presented by Temple Beth Sholom in Topeka.

The President's Message

THE LEGISLATIVE COMMITTEE is the most hardworking, diligent, dedicated committee in the Kansas Medical Society. They meet weekly when the legislature is in session (January-April) to analyze decisions and take legislative action on matters that would affect the health of the citizens of Kansas. They attempt to educate the legislators on what the medical profession thinks would be of benefit to the people of Kansas. The Committee has advance knowledge of some issues and are able to present testimony reflecting the Society's viewpoint; other issues arise with such rapidity that testimony must be formulated on the spot.



The members of the Committee are drawn from the various county medical societies in an attempt to represent viewpoints of large, small, rural, and urban areas. The first chairman of this Committee was John Blank of Hutchinson; he was succeeded by Tom Gray of Wichita, who relinquished the job to Ted Young of Topeka. Presently the Committee is chaired by Jimmie Gleason of Topeka. All have done an outstanding job.

The members of the Committee contribute time and energy which could be spent in their own practice, but they are willing to sacrifice to engage in the activities of the Committee for the benefit of quality health care in Kansas. The compensation they receive is the feeling that they have done a good and needed job. Their only reward for this—reappointment to next year's Committee.

Now that you understand the composition, the function and the endeavor of the Legislative Committee, they would appreciate your input as to how to improve their output. Please address your comments to Dr. Jimmie Gleason, Chairman, Legislative Committee, Kansas Medical Society, 1300 Topeka Avenue, Topeka, KS 66612.

Fraternally yours,

John D. Huff M.D.

President



Editorial COMMENT

The System

It began when the flight was rerouted because of weather and even then we were forced to put down at a remote and meagerly lighted field. There seemed to be no identifying signs along the route to the motel where the airline arranged to put us up. It didn't matter—the room could have been any place, and the box lunch provided by the line had the same originality.

It was later, though, after what seemed only a moment's sleep, that we became conscious that the forces of evil above and below were contending for possession of our diaphragm. We must have communicated with someone for we were next aware of being wheeled into a tiled room. The cart we were on was mounted on rails which a little man was methodically and repetitively wiping with a wad of paper toweling. We waited for a time in increasing discomfort but no one else appeared, and we were about to struggle up to ask the little man for help when the cart began to move silently through a dim tunnel.

In a moment we emerged into a brightly lighted room where multiple lights flashed intermittently on a large console closely attended by several technicians. Above the bank of lights were the letters, "VE." Directly overhead was a screen. Someone placed several wired objects on our head, and through the mist of our agony we could see that wires now connected us to the panel of lights. To one side stood a white-coated individual who was apparently in charge. At least, at his signal one of the technicians pressed a button and our discomfort was momentarily forgotten as we saw maggots of light begin to contend with each other for space on the screen over our head, while the console's frenzy increased and a continuous strip of paper began to emerge from a slot beside the director of the operations. He scanned the paper for a few moments, appeared to engage in some self-debate, then shrugged and pressed a button whereupon the cart began to move once more as a technician deftly disengaged us from the wires. We hardly had time to ponder the whole silent event when we entered another room seemingly identical to the first except that over the lighted board the identifying letters were "ML." A similar crew performed its task of scrutinizing the lights with occasional pressing of a button or flipping of a switch. Again the one in charge scanned the emerging paper strip and, appearing somewhat irritated, pressed his button and we moved silently on as before.

It is a question whether our frustration aggravated our discomfort or distracted us from it, but before we could pursue the thought we reached another of the stations, this one identified by the letters "ME." If it seemed we were getting to know the routine, no less were our attendants (if they can be so called) for at this stop there was scarcely time for the cart to come to rest before we were connected, the director was reading the print-out, and we were moving on again.

The next stop was in a larger room with three separate light panels and a much larger staff. The routine, as it happened, proved to be the same with each of the three consoles being brought into action in order. Above all of them was the designation, "EI," while each board had its own identification—"TC," "SV," and "EQ." The rituals proved to be identical with the previous ones and each other. And again, without any human sound, we rolled on our way.

Our next stop, however, was a distinct change, for it appeared to be a private office furnished with a desk upon which sat a computer screen. The only other items to be seen were two buttons on the desk's surface. Behind the desk sat another individual whose distinguished appearance and whiteness of coat denoted a higher rank than we had yet encountered. We sensed a momentary relief as we anticipated the opportunity to make contact finally with someone and gain some relief. We reckoned without our sluggish reflexes, however, for before we could speak the man pressed one of the buttons and, moving as swiftly and silently as before, we emerged into the same tiled room where we had

started our journey. And there the same little man pursued his maintenance duties.

As the cart came to a stop, we were at last rewarded with the sound of a human voice. "That," said the little man pointing toward a door, "is the way out."

"Out!" we groaned. "But I'm dying—I need help! No one does anything but push buttons and look at lights. How can I get some care?"

The little man looked at us in some surprise. "Well, now, you must not be familiar with the System. What it amounts to is that your QQ is too low and you don't qualify for care. Perhaps if you could come back when you have something a little more er—compelling—"

"What do you mean, QQ?" we moaned.

"Why, Qualification Quotient, of course," he replied. "Apparently you don't understand the testing you have been undergoing. Some people don't since it is quite the most sophisticated and illuminating system yet devised." He pondered our agonized despair a moment, then, "It really is against regulations to hold up the track this way but things are a little light at the moment so perhaps I can explain."

He paused long enough to unwad the paper in his hand which we could see was the combined printout of our adventures. "You see, the first consideration under the System is cost control and, of course, the first question therefore is whether the case is even worth bothering with—whether, to put it bluntly, the patient as an individual is worth salvaging regardless of the medical aspect. As it happens, you did pass that qualification—not too much to spare, I'll admit, but you made it."

"But how could you make any kind of a decision?" we gasped between the internal knife-thrusts. "No one said anything to me."

"Oh, that's the beauty of the System," he said. "The screen overhead presented a series of subliminal questions. You weren't aware of them—saves a lot of arguing, you know—but your brain picked them up and the answers were picked up by the electrodes and carried to the computer. These were checked there for fraud, prevarication or repetition, and stored on your master tape. If you hadn't passed that initial investigation, you would have been back here next stop."

"Great," we said in resignation, "but what was the VE for?"

"That stands for Valuation Estimate," was the reply, "a balance of your desirable features against your undesirable ones. Since you made it past that, the next stop was the medico-legal check-up—ML, you know."

"But medico-legal—", we interjected, "I'm not going to sue anyone. I hadn't even had any care to sue them about."

"Oh, but we can't be too careful," said the little man. "This measures the liability potential of the diagnosis and therapy as well as the individual. Unfortunately you didn't come out too well on this. Regardless of other things, you are a transient, you know, and that automatically puts you in a poor risk group. Anyway, you still did get by, which is why you were passed on to Economic Index."

"EI?" we asked.

"EI," he said.

"Oh," we said.

"This is, of course, the most important part of the investigation and therefore the most complicated. First came the matter of calculating the cost of therapy—CT, you'll recall. In short, the only thing that got you further consideration there was the fact that you have insurance. You got a good rating on CT since the System would be remunerated for your care. But you must realize that the economics of health care is much more complicated. Even though your VE, ML, ME and CT were adequate to get you by, your social value—SV—had to be measured. It is necessary to know not only that your expenses would be covered but that you would be productive after your recovery and not a burden on society. The System thinks of everything."

"So what happened?" we moaned.

The little man studied the crumpled paper. "Yes—well—um. Well, it seems the EQ got you. You see, under the System, we are given a quota of economic activity we can generate each month. The point is that if some control isn't put on health care costs, their part of the gross national product will go higher than the government thinks proper. While your care wouldn't actually have put us over our quota for the month, we have to save some room for the really important things—well, maybe that's not a good way to put it but after all, society can't be expected to absorb the economic impact when you know you should stay away from little green onions anyway. You get the idea."

"Only too well," we muttered in a wave of self-pity, "and that seems to be all I'll get. I didn't even get to see a doctor. No one so much as said they were sorry."

The little man looked pensive. "Oh," he said, "I'm the doctor. Sorry."

* * *

Somehow, we were back in our room and, dragging ourself to the bathroom, we surrendered to Nature's oldest therapies. Toward dawn, survival seemed assured and, despite a certain dolor of spirit, we were able to board the bus that returned us to our plane. As it took off in the lifting fog, we could faintly make out the name on the airport building. Some place called Triage.—*D. E. G.*

AMA House of Delegates

Summary of Actions Taken at the Interim Meeting, Chicago, Illinois, December 4-7, 1977

Your representatives at the AMA meeting had an interesting, but busy few days. Those officially representing the Society are the President, two delegates and their alternates, and the Executive Director of the Kansas Medical Society. Those representing you at the interim AMA meeting in early December were: John D. Huff, President; Clair C. Conard, Dodge City, and Alex Scott, Junction City, delegates; and Kermit G. Wedel and Lew W. Purinton, alternate delegates. Jerry Slaughter represented the Executive Office in Topeka.

Some two weeks prior to the meeting, we all received the AMA House of Delegates Handbook, some 2 inches thick, containing resolutions, reports of the Board of Trustees, reports of AMA councils, and other useful information. We were assigned various parts of this Handbook to review prior to the meeting. As a group, we met and reviewed the resolutions that were to be introduced and the various reports. The House of Delegates met on Sunday afternoon and the order of business consisted of awards presentations, remarks by the speaker, an address by Dr. John Budd, AMA President, and remarks by the AMA Auxiliary President. This year the House was honored to hear from Mrs. Chester L. Young of Kansas City, Kansas, who is the present AMA Auxiliary President. We were all proud to have a Kansan at the head of our Auxiliary.

Reference committees met on Monday morning. Your representatives attend these meetings in a predetermined manner. The remarkable ability of American physicians can be witnessed at the reference committee meetings; eloquent and well informed presentations are heard, both pro and con, on the multitude of problems introduced at these meetings. Since eight reference committees meet simultaneously, it is difficult for Kansas to be represented at all the meetings; however, we usually recruit enough people to make a reasonable showing. Each morning during the meeting we caucus with the Oklahoma delegation for continental breakfast, and in a preassigned manner go over the important issues to be brought before the House during the session. We expect to include the Missouri delegates at our next AMA meeting and, hopefully, we will develop some clout by combining our forces.

Customarily, the House commences its debate on Tuesday, and the order of business at the interim session is concluded by Wednesday afternoon. Probably the most important piece of business considered at this interim session was the elimination of delegates to the House from the 28 section councils, and the replacement of the section council delegates by specialty society delegate representation. The 48 specialty societies now making up the section councils may apply for delegate representation with one delegate and one alternate from each society and be seated as delegates from the specialty societies at the next interim meeting in December 1978. This would increase from 11 per cent (28 delegates from section councils) to 17 per cent (48 specialty societies) the specialty society representation in the House of Delegates. Trustee Joseph F. Boyle, M.D., of California, told the House of Delegates that "no other organization is as capable of providing the strength of leadership as the AMA. Specialty societies do want to become active participants. But they are quite ready to turn elsewhere and in some instances are." He told the House that it had a real chance to accomplish the long time objective of a strong federation for American medicine. Two sets of criteria were developed for determining how specialty societies can qualify for direct representation in the AMA House: (1) Any national medical specialty society that is currently represented on the AMA section council and which has 1,000 or more AMA members; or (2) Any national medical specialty society that is currently represented on the AMA section council and which has fewer than 1,000 AMA members, but which represents a specialty that has an approved examining board listed in the *Directory of Accredited Residencies,* published by the Liaison Committee on Graduate Medical Education. Each specialty society that meets either of these criteria would be entitled to one delegate and one alternate. Specialty societies applying after the next interim meeting may be granted representation in the House upon approval by both the Board of Trustees and the House.

The 48 specialty societies that now meet these criteria are:

American Academy of Allergy
American College of Allergists
American Society of Anesthesiologists
American College of Cardiology
American Society for Clinical Pharmacology and Therapeutics
American Society of Colon and Rectal Surgery
American Academy of Dermatology
Society for Investigative Dermatology, Inc.
American Association for Thoracic Surgery
American College of Chest Physicians
American Thoracic Society
American College of Emergency Physicians
American Academy of Family Physicians
Association of Military Surgeons of the U. S.
Society of Medical Consultants to Armed Forces
American College of Gastroenterology
American Gastroenterological Association
American College of Surgeons
American Society of Abdominal Surgeons
International College of Surgeons
American College of Physicians
American Society of Internal Medicine
American Association of Neurological Surgeons
American Academy of Neurology
The Society of Nuclear Medicine
American College of Obstetricians and Gynecologists
American Academy of Ophthalmology and Otolaryngology
American Association of Ophthalmology
Contact Lens Association of Ophthalmologists, Inc.
American Academy of Orthopaedic Surgeons
American Laryngological, Rhinological and Otological Society
American Association of Pathologists and Bacteriologists
College of American Pathologists
International Academy of Pathology
American Academy of Pediatrics
American Academy of Physical Medicine and Rehabilitation
American Academy of Facial Plastic and Reconstructive Surgery
American Society of Plastic and Reconstructive Surgeons
American Academy of Occupational Medicine
American Occupational Medical Association
American Association of Public Health Physicians
American College of Preventive Medicine
Aerospace Medical Association
American Psychiatric Association
American College of Radiology
Radiological Society of North America
American Roentgen Ray Society
American Urological Association

By margin 4 : 1, the House of Delegates renewed its support for the AMA sponsored legislation on comprehensive health insurance. In general, the consensus of the House was that the bill should be kept in and not withdrawn, but not pushed either. We were told that to have a bill in the hopper would increase chances for organized medicine to discuss the issues at the time of congressional hearings.

Action by the House will leave staff privilege decisions up to the individual hospital. According to the adopted recommendations, the determination of which physicians and other classes of health care practitioners shall be granted clinical privileges in the hospital shall be established at each particular hospital in accordance with community needs and applicable state laws and regulations. The hospital governing boards and organized medical staff were urged to consult with local legal authorities in order to keep apprised of state laws and regulations governing the extension of hospital practice privileges and limitations on the scope of practice of individuals licensed or authorized to assume direct and independent patient care services.

After some debate, the House urged that the penalties for possession of small amounts of marihuana be modified although criminal sanctions should still be imposed for drug trafficking. The House voted to support the position that, "for healthy users intermittent ingestion of even relatively potent marihuana rarely constitutes a health hazard," but warned that "regular ingestion or multiple drug use might."

The cost of health care in the United States continues to be a big issue and a resolution presented by the Student Business Session recommended that the AMA make medical students more aware of the cost of health care. Accordingly, the medical schools will be encouraged to institute educational programs aimed at increasing cost-consciousness among medical students, housestaff, and faculty. We were told that the total health care bill in the United States had increased from $42 billion to $140 billion during the last ten years, and now comprises 8.6 per cent of the gross national product.

Considerable debate resulted over a summary report of December 19, 1977, entitled, "National Commission on the Cost of Medical Care." The commission is composed of 27 representatives of business, labor, govern-

ment, consumer groups, academia, physician organizations, hospitals, and insurers. The Commission came up with 48 recommendations, some of which were in variance with, if not actually contrary to, established AMA policy. The House agreed to delay debate on this controversial report until the annual meeting at St. Louis in July, 1978, so as to provide state, county, and specialty societies an opportunity to thoroughly discuss this report. This may well be the highlight of the next AMA meeting.

CLAIR C. CONARD, M.D., *AMA Delegate*
ALEX SCOTT, M.D., *AMA Delegate*
LEW W. PURINTON, M.D., *AMA Alternate*
KERMIT G. WEDEL, M.D., *AMA Alternate*

NEW MEMBERS

The JOURNAL *takes this opportunity to welcome these new members into the Kansas Medical Society.*

Richard B. Baker, Jr., M.D.
1133 College Avenue
Manhattan, KS 66502

David J. Brown, M.D.
425 E. Murdock
Wichita, KS 67214

Michael Caughron, M.D.
5105 Neosho
Shawnee Mission, KS 66205

Deanna K. Fox, M.D.
University of Kansas Medical Center
39th & Rainbow Blvd.
Kansas City, KS 66103

Francisco Gonzalez, M.D.
1035 North Emporia
Wichita, KS 67214

Ramon R. Hizon, M.D.
929 North St. Francis
Wichita, KS 67214

James A. Littell, M.D.
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Michael B. Lasley, M.D.
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Jesus Zornoza, M.D.
University of Kansas Medical Center
39th & Rainbow Blvd.
Kansas City, KS 66103

"THE KANSAS DOCTOR"

The Kansas Doctor is available! This book is a delightful historical account of medicine in Kansas. Orders are being taken by the AMA-ERF chairmen.

Mary Ruth Yulich
2106 Chester Ct.
Kansas City, KS 66106

OR

Caryl Bichlmeier
6110 West 65th St.
Mission, KS 66202

($10.65 per copy includes sales tax and shipping.)

Month in Washington

The federal government plans a major campaign to urge the American public to seek opinions on surgery.

The unusual and precedent-setting program involving patients' dealings with physicians will be conducted by the HEW Department. Both Medicare and Medicaid programs will be geared to encourage second opinions.

The policy was announced by Hale Champion, HEW Under Secretary, before the House Commerce Subcommittee on Oversight and Investigations. The subcommittee, headed by Rep. John Moss (D-Calif.), has been holding hearings and issuing reports charging there is much unnecessary surgery in the United States.

The Department has been instructed to remove the remaining legal barriers to patient-elected second opinions in Medicare. States will be requested to implement as quickly as possible active second opinion programs for Medicaid.

If two physicians disagree, Medicare will pay for a third opinion if the patient desires one, according to HEW.

At present, Medicare will pay for a second surgical opinion if the physician agrees to the advisory or orders it. But the physician's acceptance is at present mandatory. In the future, the patient would be reimbursed for a second opinion if the initial physician believes it unnecessary.

One question to be answered is whether the patient must receive a negative response on a second opinion from the first physician, or simply could go off on his own to get a second opinion without even asking the physician.

Rep. Tim Lee Carter, M.D. (R-Ky.), has introduced legislation sought by the AMA dealing with funding for residencies in preventive medicine and labelling of prescription drug containers.

The labelling bill would require that drug containers as dispensed to patients carry the established or trade name together with the quantity and strength of the drug. The AMA said that in cases of medical emergency it is often important for attending medical personnel to know the name, strength, and contents of any drugs a patient is taking.

Under the bill introduced by Dr. Carter, ranking GOP member of the House Commerce Subcommittee on Health, an exception to the labelling is provided when the physician decides that for medical or emotional reasons it is in the best interest of the patient that the information not be made known to him or indirectly to the patient's family or associates.

The other bill introduced by Rep. Carter calls for an amendment to the Health Manpower Law to provide funding for residencies in preventive medicine. Specific program funding for such residencies was not included in the Health Manpower Law as passed.

The AMA said these residency programs are very dependent on outside funding because they generate little patient income to support their activities. The increased focus on preventive medical care makes it important that these residency programs continue, according to the AMA. The bill would provide federal funds for approved residency programs in preventive medicine and would also provide traineeships for those physicians participating.

The FDA Commissioner has stung health food advocates in an interview in *U. S. News & World Report.* In reply to a question whether health foods are safer, due to the absence of food additives, than regular supermarket products, Donald Kennedy, Ph.D., replied:

"There's not a whit of logic in that. Even if you assume that food additives are generally bad for you, it doesn't follow that their absence somehow confers safety.

"Aflatoxin, a mold product that grows on corn and peanuts, is as natural as can be and about the worst carcinogen we know," the Commissioner said.

"The 'natural' foods often cost more, but have no benefit that we can see over foods available in the regular market place."

In an attempt to do something about that spiraling health care inflation noted by Rep. Rostenkowski, three major health organizations have agreed on a sweeping national program to curb hospital rate increases through a private sector review system aimed at encouraging hospitals to seek efficiencies and to spotlight institutions that fall down on the job.

Responding to a challenge from Congress for a voluntary alternative to the Administration's proposed Hospital Cost Containment Act for federal controls, the

National Steering Committee has issued a 15-point program featuring a goal of a two per cent reduction a year in the rate of increase in hospital costs.

The steering committee was formed by the American Medical Association, the American Hospital Association (AHA), and the Federation of American Hospitals (FAH). In addition to officials of these organizations, members of the committee include officials of the Health Insurance Association of America, the Health Industry Manufacturers Association, the Blue Cross Association, consumer consultant Virginia Knauer, and the U. S. Chamber of Commerce.

At a news conference in Washington, D. C., the members of the National Steering Committee announced their agreement on the 15-point program and urged everyone involved including the public, labor, management, and the government to cooperate in the attempt to brake the rise in health care spending.

Robert B. Hunter, M.D., Chairman of the AMA Board of Trustees and a member of the committee, told the news conference that physicians and hospital personnel share the public's concern over the cost problem. "We believe the problem can be solved voluntarily better than by government intervention."

Terming the meeting "historic," Michael Bromberg, Executive Director of the FAH, said the nation's hospitals will be reviewed openly and the identity of hospitals that are overspending will be made public. "In effect this will be peer review out in the open," he said.

Under the program, each state will have a steering committee composed much like the national steering group which will receive and review monthly data from hospitals on their cost-efficiency progress.

A "very realistic goal" of a 2 per cent reduction annually in the rate of increase over the next two years was set forth by John Alexander McMahon, AHA President. This would slow the rate of increase from the current 13.7 per cent to 11.7 per cent next year, and about 9.7 per cent the following year, a level near that of the rest of the economy.

Describing the project as "a more concerted effort than any undertaken before," James H. Sammons, M.D., Executive Vice President of the AMA, said one of the key programs will be to expand public awareness of the need for cost constraints and cost awareness on the part of consumers as well as providers.

The tentative 15-point program agreed to by the National Steering Committee includes the following:

1. Creation by state hospital and medical organizations of state level voluntary cost containment committees to develop special action programs for their states;

2. Immediate reassessment by all institutions of planned budget and charge adjustments to determine what can be done to shave costs in the short run consistent with sound medical practice;

3. Make the overall national goal a 2 per cent annual drop in the rate of expenditure hikes for the next two years;

4. Set up guidelines for consideration by hospitals and state committees to identify hospitals where special efforts need to be made to cut costs. Under these guidelines, the top 15 per cent of hospitals projecting the highest increases would be reviewed first, as well as others showing a higher than average rise in expenditures;

5. As a national goal, reduce significantly the rate of the new capital investment by hospitals over the next two years. Also as a national goal—no net increase in the national total of hospital beds with certain exceptions;

6. Request that all hospital medical staffs consider ways to further tighten utilization review—consistent with sound medical practice;

7. Study and development by state committees of programs to improve productivity in hospitals by 2 per cent a year;

8. Accelerate current trends to improve the health delivery system through multi-hospital systems, shared services, health maintenance organizations, and single- and multi-specialty medical groups;

9. Notify all concerned of the national program and urge widest support and cooperation;

10. Provision of technical assistance programs by AMA, AHA and FAH to assist the state committees and hospitals in carrying out the program;

11. Urge hospital suppliers to support the program and exercise restraint in pricing;

12. Establish a subcommittee on public education to actively involve everyone in the program and to explain it to the public;

13. Seek the support of the government;

14. Call upon insurance carriers, other purchasers of care, industry, and organized labor to examine expanded consumer cost sharing, cost effective alternatives to existing coverages, and to carefully review any substantial expansion of existing benefits;

15. Seek a review by government of the cost impact of all existing federal regulations, to be completed by the end of 1979.

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Mid-America Cancer Center Program seeking Associate Director for Cancer Outreach. Experience in clinical aspects of cancer, education, and community relations. Contact James T. Lowman, M.D., Director, Mid-America Cancer Center, KUMC, 39th and Rainbow, Kansas City, KS 66103.

HEALTH RESOURCES has immediate practice opportunities for general practice and family physicians, two physicians interested in geriatric medicine, and an ENT. Other primary care and surgical openings are available July 1. Please send CV in confidence to: Dr. Ronald Hammerle, Health Resources, Ltd., Box 12621, Kansas City, MO 64116.

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Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium Br.

Please consult complete prescribing information, a summary of which follows:

Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

ROCHE

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Manati, Puerto Rico 00701

ROCHE

For recurrent attacks of urinary tract infection in women

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Each tablet contains 160 mg trimethoprim and 800 mg sulfamethoxazole.

Just one tablet b.i.d. for 10 to 14 days

- Action at urinary/vaginal/lower bowel sites helps eliminate reservoirs of infecting organisms
- Distinctive antibacterial action plus wide spectrum helps eradicate recurrent UTI
- Low incidence of bacterial resistance in community practice
- Convenient *b.i.d.* dosage provides day-and-night antibacterial control
- Contraindicated during pregnancy and the nursing period. During therapy, maintain adequate fluid intake; perform CBC's and urinalyses with microscopic examination.

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* **It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination.** *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

The recommended quantitative disc susceptibility method (*Federal Register*, 37:20527-20529, 1972) may be used to estimate bacterial susceptibility to Bactrim. A laboratory report of "Susceptible to trimethoprim-sulfamethoxazole" indicates an infection likely to respond to Bactrim therapy. If infection is confined to the urine, "Intermediate susceptibility" also indicates a likely response. "Resistant" indicates that response is unlikely.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older:

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets*, each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets*, each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension*, containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

ROCHE

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

Please see back cover.

U. of California
San Francisco, Calif. 94143

Her next attack of cystitis may require the Bactrim™ 3-system counterattack



ROCHE

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Studies have shown that Bactrim acts against *Enterobacteriaceae* in the bowel without the emergence of resistant organisms. Thus, Bactrim reduces the risk of introital colonization by fecal uropathogens. It has *no* significant effect on other normal, necessary intestinal flora.

Bactrim fights uropathogens in the urinary tract/vaginal tract/lower intestinal tract

Please see reverse side for summary of product information.

KUMC Issue

THE Journal of the Kansas Medical Society

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The JOURNAL of the KANSAS MEDICAL SOCIETY

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Possibly Effective:

1. For the relief of symptoms associated with cerebral vascular insufficiency.
2. In peripheral vascular disease of arteriosclerosis obliterans, thromboangiitis obliterans (Buerger's Disease) and Raynaud's disease.

Final classification of the less-than-effective indications requires further investigation.

Composition: Vasodilan tablets, isoxsuprine HCl, 10 mg. and 20 mg.
Vasodilan injection, isoxsuprine HCl, 5 mg., per ml.

Dosage and Administration: Oral: 10 to 20 mg., three or four times daily.
Intramuscular: 5 to 10 mg. (1 or 2 ml.) two or three times daily. Intramuscular administration may be used initially in severe or acute conditions.

Contraindications and Cautions: There are no known contraindications to oral use when administered in recommended doses. Should not be given immediately postpartum or in the presence of arterial bleeding.
Parenteral administration is not recommended in the presence of hypotension or tachycardia.
Intravenous administration should not be given because of increased likelihood of side effects.

Adverse Reactions: On rare occasions oral administration of the drug has been associated in time with the occurrence of hypotension, tachycardia, nausea, vomiting, dizziness, abdominal distress, and severe rash. If rash appears the drug should be discontinued.
Although available evidence suggests a temporal association of these reactions with isoxsuprine, a causal relationship can be neither confirmed nor refuted.
Administration of single dose of 10 mg. intramuscularly may result in hypotension and tachycardia. These symptoms are more pronounced in higher doses. For these reasons single intramuscular doses exceeding 10 mg. are not recommended. Repeated administration of 5 to 10 mg. intramuscularly at suitable intervals may be employed.

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Precautions: Do not administer more frequently than every 6 hours, or within 12 hours after rectal dose of any preparation containing theophylline or aminophylline. Do not give other xanthine derivatives concurrently. Use in case of pregnancy only when clearly needed.

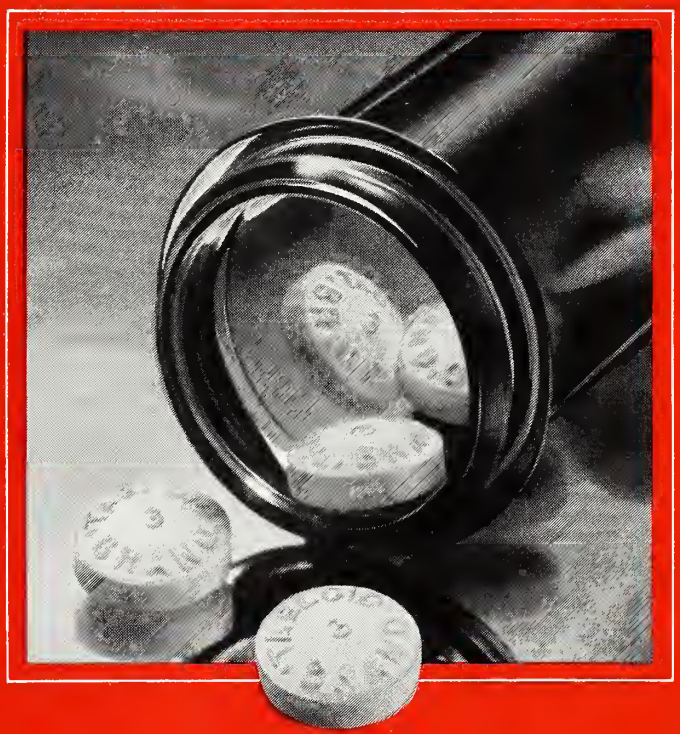
Adverse Reactions: Theophylline may exert some stimulating effect on the central nervous system. Its administration may cause local irritation of the gastric mucosa, with possible gastric discomfort, nausea and vomiting. The frequency of adverse reactions is related to the serum theophylline level and are not usually a problem at serum theophylline levels below 20μg/ml.

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This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** When the combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome. Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium sparing action of triamterene is warranted. (See Box Warning.) Routine use of diuretics in healthy pregnant women is inappropriate; they are indicated in pregnancy only when edema is due to pathological causes.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis.

'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

Supplied: Bottles of 100 and 1000 capsules; Single Unit Packages of 100 (intended for institutional use only).

FOR LONG-TERM CONTROL OF HYPERTENSION.* SERUM K^+ AND BUN SHOULD BE CHECKED PERIODICALLY. (SEE WARNINGS SECTION.)

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KANSAS MEDICAL SOCIETY

May 7-10, 1978
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Auxiliary News

A sensitive approach to the plight of the impaired physician highlighted the advent of the KMS Physician Performance Committee at the conference held in Topeka on January 26. Dr. Herbert Modlin stated that a professional is responsible to his patients and accountable to his own conscience. Willingness to police the profession is the essence of professionalism. Dr. George Penn, Chairman of the Committee, described the characteristics of chemical dependency. In his evening address, Dr. Doug Talbott of Georgia told of his state's two-year-old advocacy program that serves to identify, motivate, rehabilitate, and return physicians to the practice of their profession.

The AMA "Synergy" Leadership Conference, held in Chicago January 26-29, was affected by the big blizzard. Newscasts did not exaggerate the storm's severity. Our plane circled O'Hare Airport for an hour and a half prior to landing safely just before the airport closed. Many conference participants and faculty never arrived, but Mrs. William Crouch, KMSA President-Elect, came in on the following day after an overnight delay in St. Louis.

AMA staff improvised effectively to keep the program running smoothly. The term "synergy" was utilized to connote joint effort toward problem-solving by all segments of medicine.

The course on public relations emphasized that lack of public awareness of the facts about various problems of the health care system is a challenge that the AMA must face. It is essential to state facts, fairly evaluated, and with positive impact. Today's patient, the consumer, tends to shop around, question cost, and seek redress in in the courts. The bond between doctor and patient is not as strong as it once was.

The AMA National Advertising Campaign, to be conducted from March to November, was reviewed. This advertising will appear in the printed media which has been determined to be one of the best and least expensive procedures for dissemination of information.

The predominant topic of discussion at the Leadership Conference was health care costs, with physicians showing great concern about the actual causes and the public concept of them.

On Saturday, U. S. Senators Richard Schwieker (R-PA) and Edward Kennedy (D-MA) presented their views on the progress of health care legislation. They did not concur with the time table of the Administration's National Health Insurance proposal to Congress. During the question and answer period, a physician remarked to Senator Kennedy that most of the problems enumerated were social, not medical problems. (This listener felt that the Senator had only limited knowledge of the profession, its goals and needs, and seemed unaware that Family Practice is a specialty. Efforts by Dr. James Sammons to enlighten the Senator were given little attention.)

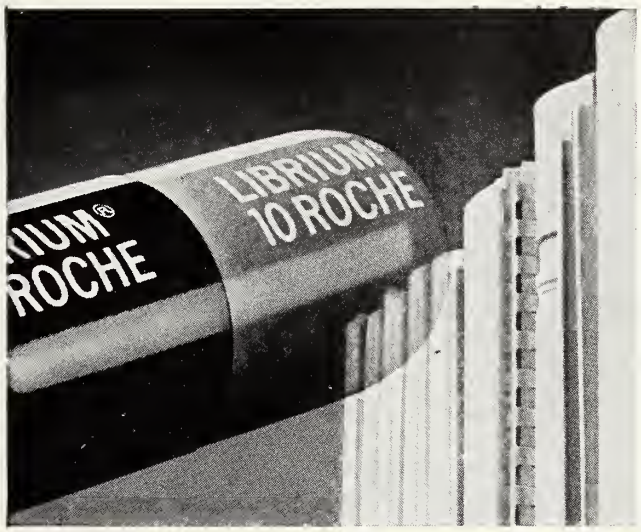
Preceding the introduction of Senator Kennedy, Dr. Sammons introduced the AMA Auxiliary President, Mrs. Chester Young, and President-Elect, Mrs. Manuel Bergnes. He recognized the Auxiliary's contribution of over $1.5 million to benefit medical education and to guarantee student loans.

The final day of the conference began with small group breakfast meetings, each led by a television or radio news commentator. Peter Hackes, Wilson Hall, Robert Mooney, and Bill Kurtis (a native Kansan) moderated the sessions and reported to the general session on physician responses to the questions, "Can there be too many physicians?" "Is regionalization the new franchise of medical practice?" "Is there unnecessary surgery?" and "What is the role of consumers in health planning and decision making?" These four gentlemen agreed that physicians should utilize the media to educate people and that the media has misrepresented the medical position. They admonished physicians to: (1) talk to the media; (2) be prepared with positive information stated succinctly; and (3) speak to local clubs—they enjoy hearing from physicians.

Bob Richards, Olympic champion and minister, challenged the conferees with a positive and dynamic address entitled "The Best Is Yet to Be."

Sincerely,
Jackie Burnett
President
Kansas Medical Society Auxiliary

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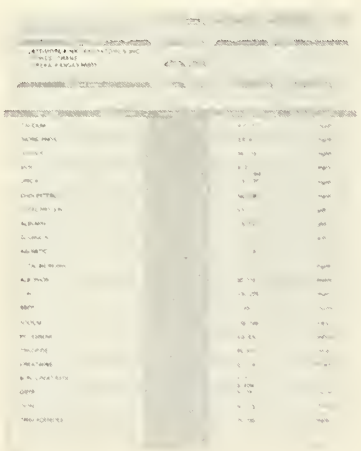
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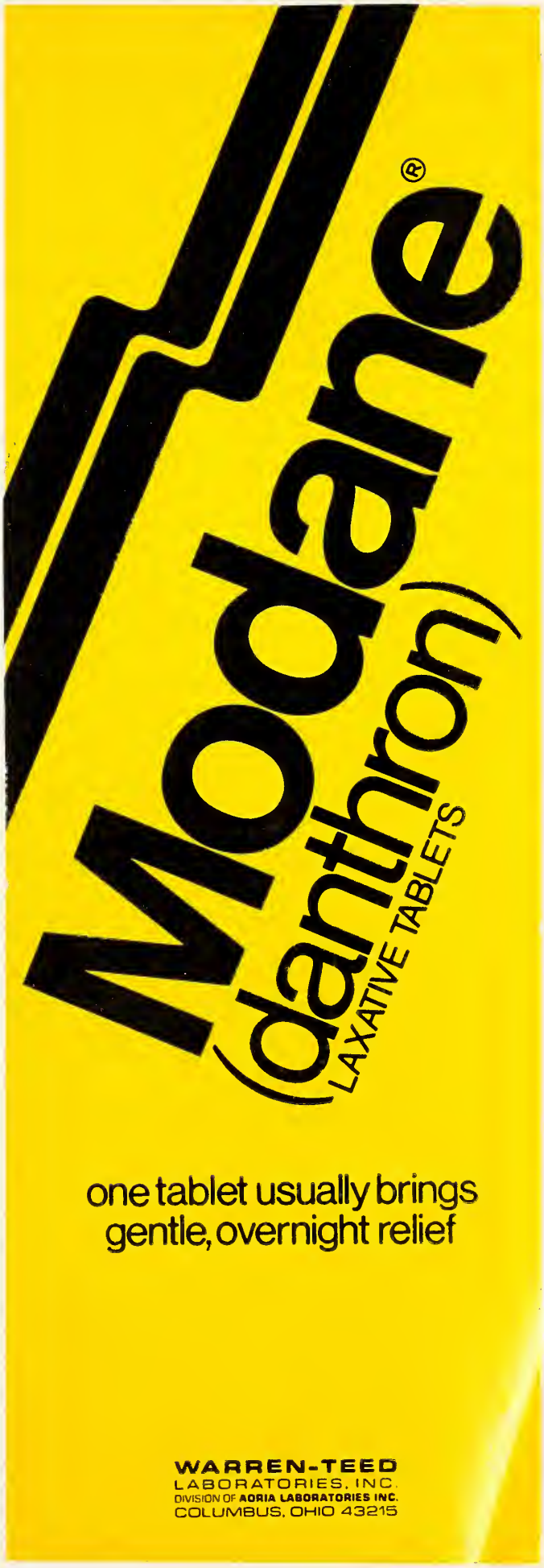
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Month in Washington

Cost of Health Care

The cost of health care has risen for the population as a whole from 6.2 per cent of the Gross National Product in 1967 to 8.6 per cent of the GNP in 1976. During these same years the cost for a semi-private hospital room rose 169 per cent and operating room costs rose 175 per cent.

According to HEW's Annual Report on Health, life expectancy in the United States has continued to lengthen and is now at a new high of 72.5 years for those born in 1975. Life expectancy for those over 65 years has also increased, climbing 2.2 years since 1950.

HEW reports that 29 per cent of the Nation's health care expenditures in 1976 were for treating those over 65. Per capita annual expense in this age group was $1,521.

The share of public funding for health care in the elderly has risen from 30 per cent in 1966 to 68 per cent in 1976, and the number of beds in nursing homes tripled between 1963 and 1973.

Between 1950 and 1974, the number of physicians in the United States rose 70 per cent from 232,697 to 394,448. The ratio of physicians to population increased 22 per cent in this period from 14.9 : 1,000 to 18.2 : 1,000.

Physician visits/person/year was 5.0 in 1973, and 4.9 in 1976. The average length of a hospital stay was 8.1 days in 1973, and 7.9 days in 1976. The percentage of persons with one or more hospitalizations in one year was 10.7 per cent in 1973 and 1976. Hospital discharges per 100 persons/year totaled 13.9 in 1973, and 14.1 in 1976.

The report also noted the rates of immunization among American children. In 1975, 32 per cent of children aged 1-4 years were not protected against measles, 38 per cent were not protected against rubella, and 35 per cent had no protection against polio.

Surgical Criteria

Under a HEW contract, the American Association of Professional Standards Review Organizations has identified 11 surgical procedures which it says "have a significant potential for inappropriate utilization."

The Association's national council has adopted a set of screening criteria for these procedures which will be sent to local PSROs. The surgical criteria "must not be viewed by local PSROs as mandating national standards," says the Chairman of the Association's Surgical Criteria Committee, John Bussman, M.D., of Portland.

Rather, the local PSRO "may wish to adopt or adapt the screening criteria for local use." In a letter to the Association's national council—"our committee has learned through experience and communications with PSROs across the country, that the 11 procedures have a significant potential for inappropriate utilization," Dr. Bussman said.

The 11 surgical procedures are:

> abdominal hysterectomy, vaginal hysterectomy, coronary arteriography, cataract removal, dilation and curettage, tonsillectomy and adenoidectomy, cholecystectomy, hiatial hernia repair, lumbar disc excision for rupture or protrusion, meniscectomy, and appendectomy.

With respect to vaginal and abdominal hysterectomy, a subject of national attention, the national council said:

"Sterilization by abdominal (or vaginal) hysterectomy is acceptable only in the presence of concomitant uterine disease."

Blood Labeling

All whole blood drawn after May 15, 1978, for transfusion must be labeled "paid" or "volunteer" donor.

The final regulation of the FDA specifies that persons who do not receive monetary payment for blood are classified as volunteers. The "volunteer" designation includes those who receive benefits other than money, such as membership in a blood assurance program or leave from work.

The labeling requirement also covers red blood cells, anti-hemophiliac factor, platelet concentrate, and single plasma.

The blood labeling rule caps a lengthy nationwide debate on national blood policy. In issuing the regulation, FDA Commissioner Donald Kennedy, Ph.D., said the labeling rule is "consistent with the goals of the government's national blood policy to move the country to an all volunteer system."

The incidence of post-transfusion hepatitis has been

(Continued on page 120)

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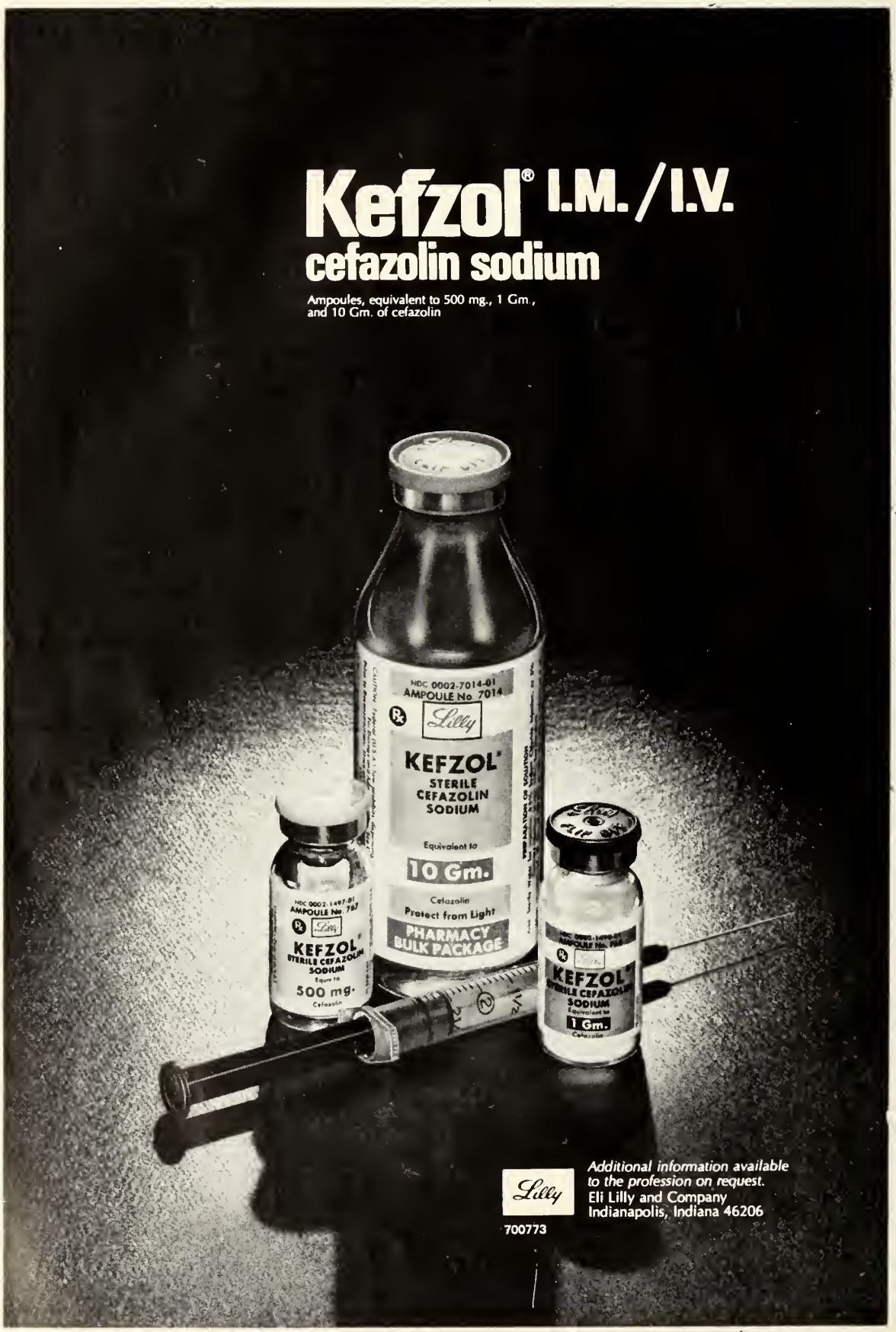


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Thirty-Second Annual University of Kansas School of Medicine Issue

The Dean's Letter—1978

The University of Kansas Medical Center

DAVID WAXMAN, M.D.,* ***Kansas City, Kansas***

I WELCOME the opportunity to address the readers of *The Journal of the Kansas Medical Society.* During the years I have been associated with the University of Kansas Medical Center, I have had the privilege of meeting many of you. In my new role, which I assumed in December, I hope to become personally acquainted with even more Kansas physicians. I need your counsel and I welcome your suggestions. Let me hear from you!

In January of this year, the Kansas Board of Regents unanimously adopted the following resolution:

WHEREAS, There is a shortage and maldistribution of physicians in Kansas, especially in the field of family practice medicine; and

WHEREAS, There is convincing evidence that there is a significant correlation between the place where a physician takes residency training and where he or she establishes medical practice; therefore be it

Resolved by the Board of Regents that the University of Kansas is encouraged to seek legislative support for the additional residency positions which the Board and the Governor have recommended so there will be available in the State of Kansas a residency position for each graduate of the University of Kansas School of Medicine; and be it further

Resolved that, to produce more family practitioners, the Board of Regents encourages the University of Kansas to proceed with plans to expand further the number of residencies in the Department of Family Practice; and over the state; and the Board of Regents urges members of the medical community and the State Legislature of Kansas to support these efforts in appropriate ways.

THIS IS OUR CHARGE: We are committed to do everything within our power to accomplish the desired results.

The columns of *The Jouranl* in recent years have carried announcements of the variety of programs the University has instigated to address the health care needs of Kansas over the long term. I will not review all of them. However, I call your attention to the following developments:

* During the past year, the College of Health Sciences has appointed two deans of the School of Medicine—James T. Lowman, M.D., in Kansas City, and Richard A. Walsh, M.D., in Wichita;
* We have established a physician placement and recruitment office. Operational for only a few months, it is already beginning to produce results. More than 100 Kansas communities are seeking assistance in the recruitment of physicians. More than 220 inquiries or expressions of interest have been received from physicians in other states in response to nationwide advertising;
* Rural Health Weekend, an effort to expose medical students to the opportunities of practice in Kansas, started last year with one weekend. This year, two are planned;
* The nurse practitioner program of our School of Nursing now has students enrolled, and the first graduate has completed requirements;
* We have established our first regional health education center concept in Kansas. The Chanute center is in the developing stages to serve Southeast Kansas, and funding has been requested to begin developing a similar center in Hays for the northwest part of the state. Such centers can serve as headquarters for a variety of medical support programs which help practicing physicians in their work. They can be the area center for continuing education programs for doctors, nurses and allied health professionals, and can serve as a location through which communication can be regularly maintained with other physicians in the area and with faculty clinicians in the School of Medicine, helping to minimize possible isolation of physicians who practice in small communities;
* Construction is underway on the Radiation Therapy Center which will bring to the state and the region a cancer treatment facility of the first order;
* This spring, the University will graduate its first 200-member class. When these students complete residency training, there will be additional physicians establishing their practices in Kansas.

I am sure all of my precedessors have had personal goals of major importance to them as individuals. I do also. My goals are to: (1) further upgrade our educational programs at all levels, *i.e.*, undergraduate, house officer, and importantly, continuing education for the

(Continued on page 129)

* The Executive Vice Chancellor, University of Kansas School of Medicine, Kansas City, KS 66103.

Developmental Disabilities Clinic

A Multidisciplinary Approach for Handicapped Children

LILLIAN GONZALEZ-PARDO, M.D.,* *Kansas City, Kansas*

A multidisciplinary approach for handicapped children seen in the Developmental Disabilities Clinic is presented, emphasizing the most common clinical problems seen in 314 children referred over a 34-month period. The role of the physician in the diagnosis and management of developmental disorders is described.

With improvement in care of acute conditions in infants and children, a growing emphasis and concern has focused on developmental disabilities; although not life threatening, they may result in serious loss of human potential. Developmental disabilities (DD) are a group of neurological handicapping conditions that include mental retardation, epilepsy, behavioral disorders, motor and learning disabilities. These disabilities originate before age 18, continue indefinitely, and constitute substantial handicaps for children and their families.

The physician who deals with children continues to be the initial source of diagnostic and therapeutic services for the developmentally disabled child and his family. The child with DD rarely has a single problem. The comprehensive diagnostic and therapeutic processes require a wide range of professional support, and a multidisciplinary team approach to these problems has evolved. The team approach to complex medical and psychosocial problems utilized in child abuse and neglect, leukemia and other malignancies, congenital heart disease and developmental disabilities are efforts to provide total care to patients and their families.

The impetus to the multidisciplinary approach was facilitated by the establishment of University Affiliated Programs for the diagnosis and treatment of the DD population as well as support for training of professionals in the area of DD.

The Children's Rehabilitation Unit/University Affiliated Facility (CRU/UAF) at the University of Kansas Medical Center College of Health Sciences and Hospital, under the Department of Pediatrics, is a multidisciplinary training facility for the diagnosis, treatment, and management of developmental disabilities. Professional staff represented are pediatrics (neurology, endocrinology, birth defects), special education, nursing, social work, psychology, speech pathology, audiology, physical therapy, occupational therapy, dentistry, and orthoptics. The different disciplines are divided into several diagnostic teams, and single or multiple disciplines are represented in several ongoing treatment groups in the unit.

Referrals are processed through a multidisciplinary intake committee that determines appropriateness and availability of resources in the unit.

The Developmental Disabilities Clinic was initiated in January 1975, primarily as a medical-neurological specialty clinic for patients seen through any of the multidisciplinary diagnostic teams or direct referrals for neurologic evaluation that may require other professional disciplines in the unit.

Patient Population

Over a 34-month period beginning in January 1975, 314 patients have been seen in the DD Clinic. Ages ranged from 2 months to 18 years; 216 patients (69%) were aged 5-15 years, 88 (25%) were under age 5 years, and 10 (6%) were over age 15 years.

Clinical Problems

The patients are divided into two major groups depending on the nature of the referring problems: Group 1 represents the school-related problems as shown in *Table I;* Group 2 includes non-school related problems as shown in *Table II.* One third of all patients had more than one problem.

School-related problems are defined as those referrals initiated by school personnel, physicians or parents that concern a child's academic performance, behavioral or emotional problems interfering with school activities, and problems that are suspected to be of medical or

* From the Children's Rehabilitation Unit/University Affiliated Facility, KUMC.

Address reprint requests to Dr. Gonzalez-Pardo, KUMC, 39th & Rainbow Blvd., Kansas City, KS 66103.

TABLE I

SCHOOL RELATED PROBLEMS

(Number of Patients = 182)
Boys = 142 Girls = 40

Diagnosis	*Frequency**	*%*
Behavior/emotional problems	56	25
Specific learning disability	45	21
Educable mental retardation	43	20
Visual, auditory, or motor problems	42	20
Convulsive disorders	24	11
Others	7	3
	217	100

* 35 patients had more than one problem.

TABLE II

NON-SCHOOL RELATED PROBLEMS

(Number of Patients = 132)
Boys = 73 Girls = 59

Diagnosis	*Frequency**	*%*
Developmental delay	78	37
Behavior/emotional problems	35	17
Convulsive disorder	31	15
Visual, auditory, or motor problems	49	23
Others	16	8
	209	100

* 77 patients had more than two problems.

neurologic basis needing evaluation to determine their effects on a child's school performance. This type of referral is assigned to the School Diagnostic Team, a multidisciplinary specialty team composed of neurology, psychology, special education, occupational therapy, orthoptics, social work, audiology, and speech pathology specialists.

Non-school related problems are those referrals requesting developmental assessments for diagnosis and management purposes and placement in programs within the unit or in other community agencies. The primary problem presented in this group does not usually involve school or academic performance. This type of referral is assigned to another multidisciplinary diagnostic team.

Of the total 314 patients, there were 182 patients (58%) whose problems were school related, and 132 (42%) whose problems were non-school related.

The sex distribution in Groups 1 and 2 shows a marked male predominance, 3 : 1 ratio in the former and 1.2 : 1 in the latter group.

The referral sources are shown in *Table III*. Eighty-five of 99 referrals from the school are school-related problems.

Seventy-six per cent of all patients seen in the DD Clinic were also seen by more than three other disciplines in the evaluation process. Psychology, special education, and audiology were involved in evaluation of 85 per cent of all school-related problems; and physical therapy, occupational therapy, psychology, audiology and speech pathology in 70 per cent of non-school related problems.

Discussion

It is estimated that there are approximately six million handicapped children in the United States today. Sixty to seventy per cent of these children can be helped to become useful to society, but fewer than half are currently getting the help they need. The plea for early recognition of developmental disabilities must reach the professionals who can influence children and their parents. Pediatricians and family practice physicians who are in this role have a major responsibility for the care of these children, and must develop an awareness of professionals and facilities in the community who may lend their support to habilitation of the handicapped child.

Because children with DD have multiple problems and parents must be provided with support and counseling in understanding their children's needs, a multidisciplinary approach is the ideal habilitation program. The physician should no longer carry the sole responsibility, but must be knowledgeable of the expertise other professionals can contribute toward diagnosis and management. Denhoff[1] clearly pointed out that the responsibilities the physician carries are tremendous and require special skills in undertaking and communicat-

TABLE III

REFERRAL SOURCES

Referral Sources	*Number*	*%*
Physicians	110	35
School personnel	99	32
Parents	31	9
Others*	75	24
	314	100

* Mental health clinics, social rehabilitation services, other professionals.

ing with other disciplines in carrying out the ultimate goal of delivery of exemplary service to these patients and their families.

School Problems

In our experience, the majority of referrals are school-related problems. The most common school-related problems referred are school underachievement, behavioral problems, and "Please rule out neurological problems." The schools are the source of referral in half of the cases, and physicians in a fourth of the cases, seen by the School Diagnostic Team. On the basis of our experience, not all school underachievement and behavior problems are due to a specific learning disability in the child.

The concept of specific learning disabilities is probably one of the most misunderstood problems in pediatrics. A good definition of specific learning disabilities is one that combines medical and the educational aspects. Kass and Myklebust[2] include the following criteria in the definition of specific learning disabilities:

1. Specific learning disability refers to one or more significant deficits in essential learning processes requiring special education techniques for remediation;

2. Children with specific learning disability generally demonstrate a discrepancy between expected and actual achievement in one or more areas such as spoken, read or written language, mathematics, and spatial orientation;

3. The learning disability referred to is not primarily the result of a visual, hearing or motor handicap, mental retardation, emotional disturbance, environmental disadvantage, or lack of opportunity to learn.

Only one fifth of our patients had the diagnosis of specific learning disability on the basis of the above criteria. It is clear, therefore, that factors other than the child, such as parent and school environment, must be taken into consideration in the assessment of school problems. Children with specific learning disabilities are of normal or average intelligence but experience significant deficits in some academic areas and thus require special remediation techniques. One fifth of the problems related to academic underachievement presented in the clinic were children in the educable mentally retarded range with intellectual abilities below normal and, therefore, experiencing academic deficits in all areas. These children still require special classrooms geared for their abilities, instead of being forced into regular classroom situations by parents who are offended by the stigma of a "special classroom."

The objective of the medical assessment is to demonstrate the existence of any causative factors of disease or injury capable of amelioration or prevention. The presence of visual, auditory, and motor factors in a child's school performance are assessed. Degenerative neurological conditions are fortunately rare, but are looked for on the basis of the clinical history, the neurological examination, and other diagnostic tests if indicated. Potentially treatable but previously unrecognized, convulsive disorders can be successfully treated with the appropriate anticonvulsant medications. After the elimination of the above conditions, however, there is usually a paucity of positive neurological findings that may or may not contribute to the child's educational program. However, it offers a relief and peace of mind to parents and referring physicians that a serious brain disorder has been ruled out.

The special education consultant and the psychologist on our team are key persons in the assessment and management of the learning disabled child who usually also has a behavior problem. The educator, on the basis of the home school's testing, determines academic skills, assesses the child's strengths and weaknesses in the academic areas, and can recommend a specific teaching technique or method that would work best for the particular child. Trial teaching is sometimes done to test out the most effective way to develop the child's abilities. Those recommendations are made available to the child's home school teacher who will eventually carry out the recommendations. It is, therefore, imperative that we work closely with the home school in our evaluations. The psychologist, on the basis of behavior observations and skills in psychological testing, is able to make positive suggestions to manage the child more appropriately both at home and in the classroom.

In the past year, 16 school visits were made by a small group of our staff in the school team to determine: (1) the exact nature of the child's problem related to the school's physical environment; (2) to evaluate the appropriateness of teaching techniques and methods and to help the school develop guidelines for the child; and (3) to evaluate behavior and interaction of the child in relation to his peers, teachers, and the rest of the physical school environment. We found the school visits extremely valuable in confirming the nature of the child's performance and behavior, and in influencing the team in making appropriate recommendations for placement and behavioral management. Together the physician, the educator, and the psychologist contributed in the interdisciplinary assessment and management of the child experiencing school failure. Counseling is offered in some cases of family problems, and because the recommendation is made in the interest of the child, the parents find counseling easier to accept. We realize that school visits cannot be done by every

physician, psychologist or special educator, but the importance of opening communications with the school personnel who are dealing directly with the child is extremely valuable in helping the child experience more success in school, and for parents to understand the nature of the child's problem.

Developmental Delay

The child with developmental delay is a common referral problem in our unit. The concept of developmental disorders encompasses a group of deficits in neurological development that result in impairments in one or a combination of skill areas such as intellectual abilities, motor deficits, language deficits, and behavioral abnormalities. With early screening and recognition of any or all of these deficits, earlier intervention can prevent more serious and complex problems for the child. Johnston[3] warns that when making an early diagnosis, labeling for the sake of a label should be avoided. The more important aspect in early diagnosis is to identify the specific skill or problem area; this is necessary before professionals can even begin a meaningful treatment program. It is now passé to say that any child is too young to be tested.

Physical and occupational therapists have made significant contributions in our team in the assessment of motor skills, as well as making appropriate recommendations for programs to help the handicapped child develop self-help skills and to achieve his full potential. Audiologists and speech pathologists assess unrecognized hearing deficits that may contribute to speech and language delay; the child with developmental aphasia, dysarthria or delay are appropriately assessed, and programs recommended for both school and home as well as individual therapy if indicated. Social work skills are utilized best in working with families whose interpersonal problems contribute to the child's problem.

Pediatric dentistry is part of the total health care program in our unit. Although some of the children have their own dentists, the child seen as handicapped because of mental retardation, epilepsy, and motor disabilities may present with unique problems.

A multidisciplinary team must be interdisciplinary in process. The successful team is one whose members have mutual trust and respect for each other. Open and shared communication of all professionals involved must exist. The physician who has traditionally enjoyed "medical elitism" is often the logical choice for leadership in the team, but not all physicians have the time, interest, or ability to carry the entire caseload of the DD population in their community. Depending on leadership abilities of those in other professional disciplines, such as nursing, psychology, physical therapy and others, they may also provide the leadership for the team. The important and critical function of the team leader is to unite and facilitate the team to a common action plan, with input from the expertise of the other professional disciplines. Local and regional DD councils can often serve as advocate for the developmentally disabled.

Although there is no dramatic cure in the treatment and management of DD, physician involvement must be encouraged as a continuing health resource for infants and children. Management and support for these children and their families are often complex, long-term, and at best ameliorative rather than curative. The philosophy in the overall approach to DD is well expressed in Sir William Osler's words, "To cure sometimes, to heal often and to comfort always."

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Mark Your Calendar Today!

119th KMS ANNUAL SESSION

May 7-10, 1978 **Ramada Inn** **Topeka**

Short Stature

Evaluation and Diagnosis Procedures at KUMC

PHILIP J. McDONALD, M.D. *and* **WAYNE V. MOORE, M.D.,** *Kansas City, Kansas*

Short stature was studied under two general headings: non-endocrine and endocrine causes. Under non-endocrine, five separate categories were distinguished, including constitutional short stature, primordial short stature, skeletal disorders, genetic short stature, and metabolic causes. A retrospective study of 161 patient charts has been reported according to the above variations, and useful comments as to the applications of these factors in the evaluation of short statured patients have been offered.

THE CHILD who is short or fails to grow adequately despite a nurturing environment often presents a diagnostic dilemma. A child who has had regular checkups and is thought to be attaining a normal height may suddenly be found to be short when compared with his peers at school. Parental anxiety and physician concern for a child's short stature may result in referral to the endocrinologist for evaluation.

This report reviews the diagnostic categories of short stature and delayed growth used in the Pediatric Endocrinology Clinic at the University of Kansas Medical Center. A summary of the results of our evaluation of these patients referred between January 1972 and May 1977, is also presented. From these data, a rational approach to evaluation of short stature can be formulated.

Methods

In order to classify the children with growth problems, it was necessary to obtain certain data. For the purpose of this study, the data were obtained from a retrospective chart review of 161 patients. The data were judged incomplete on 17 patients who were excluded from the study. The skeletal age or bone age (BA) was determined from an x-ray film of the left wrist compared to standard x-rays in the Greulich and Pyle Atlas.[1] The height of the child was obtained on a rigid measuring device (statiometer) and plotted on standard growth curves.[2] (Our growth charts are based on data from the National Center for Health Statistics; NCHS Growth Charts, 1976, are available through Ross Laboratories.) Values less than the fifth percentile were considered abnormal. The age on the growth chart at which that height intersects the 50th percentile of the standard height curve was the height age (HA) of the child. The growth rate (height velocity) expressed in cm of growth/year was determined from at least two heights plotted at six month intervals on the growth chart. Height velocities of <5 cm/yr were considered to be pathological. Children who were growing below or at the minimum of 5 cm/yr were usually admitted to KUMC for full evaluation of endocrine and metabolic functions. The projected adult height was estimated from Bayley-Pinneau Tables[3] in the Greulich and Pyle Atlas using the bone age and chronological age. Adult heights greater than 152 cm (5 ft) in females and 162.2 cm (5 ft 4 in) in males were considered normal. Birth weight and birth length were obtained by history or from hospital records. In a full term infant, a birth weight of less than 2500 gm (5 lb 10 oz) in the male or 2400 gm (5 lb 4 oz) in the female was considered abnormal. A birth length less than 46 cm (18 in) in the male and 45 cm (17¾ in) in the female was similarly abnormal. Growth records available at the initial referral were evaluated as being adequate (at least two measurements six months apart), inadequate (one point), or no information. The difference between the chronological age and bone age (CA-BA), and chronological age and height age (CA-HA) were computed.

From the above data, the patients were placed in one of the diagnostic categories.

Definition of Terms

The children with short stature were placed into different categories[4, 5] for purposes of evaluation and discussion. The first categories are listed under the broad rubric of "non-endocrine" causes of short stature. Even though endocrine and metabolic factors are likely involved in the pathogenesis of the "non-endocrine" conditions, it is not possible at this time to relate the cause

TABLE I

PATIENT POPULATION REFERRED FOR EVALUATION OF SHORT STATURE IN THE PEDIATRIC ENDOCRINOLOGY CLINIC AT KUMC—JANUARY 1972-MAY 1977

	Patients						*Referring Records*					
	COMBINED		MALE		FEMALE		NONE		INADEQUATE		ADEQUATE (NO FALLOFF)[1]		ADEQUATE (FALLOFF)[1]	
Category	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Non-endocrine														
Constitutional short stature	52	36	48	92	4	8	12	24	5	10	27	53	7	13
Familial short stature	16	11	10	63	6	37	8	50	2	12.5	4	25	2	12.5
Primordial short stature	18	12.5	11	61	7	39	9	50	1	5	5	28	3	17
Skeletal disorders	2	.01	2	100	0	—	1	50	1	50	0	—	0	—
Genetic short stature	20	14	2	10	18	90	7	35	2	10	6	30	5	25
Metabolic disease	1	.051	1	100	0	—	0	—	1	100	0	—	0	—
Endocrine														
Panhypopituitarism	6	4	3	50	3	50	1	25	0	—	1	25	2	50
hGH deficiency	17	12	14	82	3	18	4	40	1	10	1	10	4	40
Hypothyroidism	8	6	0	—	8	100	4	50	0	—	4	50	0	—
Hypoparathyroidism	1	.007	0	—	1	100	1	100	0	—	0	—	0	—
Miscellaneous														
Chronic disease, nutritional	3	1	1	67	1	33	0	—	1	33	1	33	1	33
Total	144	100	93	65	51	35	47	35	14	10	49	37	24	18

1. No falloff is taken as records indicating normal growth rate and a growth curve that parallels the normal growth curve.

of the abnormalities of short stature to identifiable endocrine gland dysfunction. The "non-endocrine" categories of short stature are:

Constitutional short stature. These children have normal birth length and birth weight. Their skeletal age is retarded and growth rate is normal. Projected adult height is normal or greater than 162.2 cm in males and 152 cm in females.

Primordial short stature. These children have parents of normal size. Bone age is not retarded. Projected adult height is less than normal. The birth length or weight of these children is less than the fifth percentile. Children may have signs of intrauterine growth retardation.

Skeletal disorders causing short stature are usually obvious from assymetry or disproportion.

Genetic short stature refers to children with identifiable syndromes or chromosomal abnormalities that have associated short stature.

Metabolic causes of short stature include those conditions with inborn errors of metabolism which result in abnormal growth or short stature. Endocrine causes of short stature include identifiable and potentially treatable deficiencies of hormones necessary for normal growth. These commonly include hypothyroidism, isolated growth hormone deficiency, panhypopituitarism, and disorders of parathyroid gland.

Results

The largest diagnostic category in our study was constitutional short stature (36%), which was comprised almost entirely of males (92%) *(Table I)*. Endocrine causes of short stature were diagnosed in 22 per cent of the cases with roughly equal distribution between male and female. Familial short stature, primordial short stature, and genetic short stature were diagnosed with roughly equal frequency. Eighteen (90%) of the genetic short statures were diagnosed as Turner's syndrome, making it the most common cause of genetic short stature and the most common cause of short stature among females.

The average ages of diagnosis did not vary significantly among the groups *(Table II)* except for skeletal disorders, which tend to be apparent at an early age. Referring information was judged adequate (two heights at least six months apart) in 55 per cent of cases, inadequate (one height) in 10 per cent of cases, and no information was available in 35 per cent of the cases. It is interesting to note that two-thirds of those

TABLE II
CHARACTERISTICS OF PATIENTS WITH SHORT STATURE

Category	Average Age at Diagnosis yr	Growth Rate cm/yr	Growth Rate (Post-treatment) cm/yr	Projected Adult Height MALE cm	Projected Adult Height FEMALE cm
Non-endocrine (n = 109)					
Constitutional short stature (n = 52)	10.1 ± 4.6[1]	5.2 ± 1.3[2]	—	166.1 ± 17.7	157.9 ± 2.7
Familial short stature (n = 16)	8.4 ± 4.2	4.4 ± 1.3	—	159.1 ± 7.4	145.8 ± 2.9
Primordial short stature (n = 18) ...	8.9 ± 4.8	4.9 ± 0.6	—	157.9 ± 5	147.1 ± 2
Skeletal disorders (n = 2)	2.0 ± 1.4	4.5	—		
Genetic short stature (n = 21)	9.3 ± 5.3	4.6 ± 1.3	—	150.7 ± 6.3	149.7 ± 6.6
Turner's (n = 18)	9.1 ± 5.3	4.6 ± 1.3	—	—	149.7 ± 6.6
Endocrine (n = 32)					
Panhypopituitarism (n = 6)	9.4 ± 5.2	3.2 ± 1.0	5.4 ± 1.6[3]	165.5	150.7
hGH deficiency (n = 17)	10.6 ± 3.9	2.5 ± 2.0	5.2 ± 2.2	161.6 ± 8	164.2
Hypothyroidism (n = 8)	9.8 ± 3.6	3.0 ± 1.4	6.9 ± 1.4	144.8 ± 6.3	144.8 ± 6.5
Hypoparathyroidism (n = 1)	14	4.0	7.3		150.8
Miscellaneous					
Chronic disease, nutritional (n = 3) .	9.3 ± 6.9	4.0 ± 1.0	8.5 ± 4.95	168.2	149.7

1. No significant differences among the groups.
2. All growth rates of non-endocrine groups differed significantly from those of endocrine group ($p < .05$).
3. All post treatment growth rates differed significantly from pre-treatment rates ($p < .05$).

referrals with adequate referral information demonstrated a short but normally growing child. One-half of the referrals of the children with endocrinological problems did not have adequate referral information.

The degree of skeletal retardation was compared among the various groups by calculating chronological age minus bone age (CA-BA) *(Table III)*. The differences between the CA-BA for the constitutional and endocrine groups, and the familial and primordial groups were statistically significant ($p < 0.05$). The Turner's group also had a significant ($p < 0.05$) delay in bone age (CA-BA) when compared with primordial and familial short statures. The largest retardation in bone age was observed in the hGH deficient and hypothyroid groups, which was further delayed by about 1 year when compared to the constitutional group.

A comparison of the bone age to height age (BA-HA) demonstrated the largest differences in the familial, primordial and genetic short statures. The BA-HA for these groups were significantly ($p < 0.05$) greater when compared to those of the constitutional short statures (0.23 year), or the endocrine group (.85-1.26 years). The BA-HA in the constitutional group was also significantly ($p < 0.05$) less than those in the endocrine group.

A comparison of the chronological age to height age (CA-HA) demonstrated that the CA-HA of the "non-endocrine" group (2.58-2.86 years) was significantly ($p < 0.05$) different from that of the endocrine group (3.8-4.38 years) *(Table III)*. Computation of CA-HA/CA was significant only for the constitutional group when compared to the endocrine group, and was not considered to add any additional information.

Discussion

The information obtained from this study can be used for developing a rational approach to the evaluation and diagnosis of growth failure and short stature. Generally the patients can be divided into two groups, endocrine and non-endocrine, based on the growth rates. Those individuals with growth rates of <5 cm/yr are most likely to have a definable endocrinological or chronic disease as a cause for their short stature. In fact, any child with a growth rate of <5 cm/yr as demonstrated by a "fall-off" from the normal growth curve deserves a prudent and expeditious evaluation of the failure to grow. The most common endocrinological cause of short stature was isolated (usually idiopathic) hGH deficiency in males and hypothyroidism in females. Fortunately, growth hormone and thyroid replacement are available for treatment of these conditions; however, if diagnosis and treatment are delayed, the likelihood of attaining a normal adult height

TABLE III
DIAGNOSTIC INDICES OF SHORT STATURE INDEX IN YEARS

Category	*CA-BA*	*BA-HA*	*CA-HA*	*CA-HA/CA*
Non-endocrine				
Constitutional short stature	2.6 ± 1.0	0.2 ± 0.9	2.7 ± 1.2[3]	.32 ± .14[4]
Familial short stature	1.1 ± 1.4[1]	1.8 ± 1.1[2]	2.6 ± 1.3	.34 ± .15
Primordial short stature	1.3 ± 1.1	1.6 ± 1.5	2.9 ± 1.4	.37 ± .12
Genetic short stature (incl. Turner's)	2.0 ± 1.6	1.4 ± 1.3	2.8 ± 1.9	.33 ± .14
Turner's	2.3 ± 1.6	1.1 ± 1.1	2.9 ± 1.9	.35 ± .12
Endocrine				
Panhypopituitarism	2.8 ± 1.0	1.3 ± 1.0	4.0 ± 1.8	.46 ± .14
hGH deficiency	3.7 ± 1.5	1.9 ± 1.6	4.4 ± 2.2	.42 ± .15
Hypothyroidism	3.3 ± 1.8	0.9 ± 1.2	3.8 ± 1.4	.41 ± .12
Miscellaneous				
Chronic disease, nutritional	2.8 ± 2.1	1.2 ± 1.3	3.3 ± 1.6	.44 ± .13

1. Familial and primordial CA-BA differed significantly from all other groups ($p < .05$).
2. BA-HA for familial, primordial and genetic all differed significantly from constitutional and endocrine groups ($p < .05$).
3. All values in non-endocrine group differed significantly from all endocrine values ($p < .05$).
4. No significant differences except between constitutional and endocrine groups ($p > .05$).

is decreased, as reflected in the lower than normal projected adult heights in these groups. Our center currently treats 22 children by hGH therapy provided through the National Pituitary Agency, but we can treat these children only until they reach a height of 162.2 cm.

An accurate assessment of a child's growth rate usually requires a minimum of two measurements taken at least six months apart. Ideally, the child should be weighed and measured and the information plotted on a growth curve at each visit to the doctor's office. This simple procedure will provide longitudinal information about the child's growth that is most helpful in detecting early growth failure. This should allow evaluation and diagnosis prior to the time when the growth failure results in a child who is significantly shorter than the peers and less than the third percentile for height. Early diagnosis and treatment is more likely to result in attainment of normal adult height.

Categorization of children in the non-endocrine causes of short stature can usually be accomplished with relatively few tests, assuming that longitudinal growth data indicates growth that parallels the normal growth curve with growth rates of at least 5 cm/yr. The constitutional, familial, and primordial short statures can be distinguished on the basis of history, BA, CA-BA, and BA-HA. Constitutional short statures will exhibit bone ages significantly retarded from their chronological age (CA-BA >1.5) and bone ages roughly equal to the height age (BA-HA = 0). These children will experience delayed puberty and will have normal projected adult heights. Familial short statures will have a family history of short stature and their bone ages will be roughly equal to their chronological ages (CA-BA = 0) and their bone ages will be significantly above their height ages (BA-HA >1). Primordial short statures will have a CA-BA and BA-HA similar to that of the familial short statures, but will have a birth length or birth weight less than the third percentile. These guidelines state in more specific terms what should be intuitively obvious, that a retarded bone age suggests that significant growth potential remains. This is reflected in the normal projected adult heights in the constitutional short statures, but abnormal projected adult heights for the familial and primordial short statures.

It was also obvious during the study that the classification into the familial, primordial, and constitutional categories was often arbitrary since significant overlap occurred among the groups. This was apparent from the overlap of the different parameters used to classify the children *(Tables I-III)*. For example, it was not unusual to identify children with familial short stature who have a constitutional or primordial component. All possible combinations tended to exist within these groups and occasionally with other groups as well.

In terms of management, the thoughtful physician can do much to prevent the psychological scarring of people with short stature. With constitutional short stature, the assurance that the child and the projected

adult height are normal is usually sufficient to allay the parental and patient anxiety over the short stature or delayed puberty. If these males with constitutional short stature have not entered into puberty by the age of 14½ to 15 years, it may be helpful to initiate pubertal development with 200 mg depo-testosterone/month for 3-6 months. This treatment solves the psychosocial problems associated with absent secondary sexual development at this age. Once initiated, the secondary sexual development usually continues by normal mechanism.

The management of patients with primordial or familial short stature involves counseling regarding the expectations of a child with short stature. This counseling should aim to achieve at least the following: (1) personal expectations and interactions of the child consistent with the child's chronological age rather than the height age; (2) if the child insists on competitive athletics, channeling of the child's activities either to competition that is with others of equivalent size or competition where size is not a major determinant in the ability to compete; (3) elimination of factors that might further limit the child's ultimate height potential; and (4) psychological adjustment to the short stature by the family and child to prevent it from becoming a hindrance.

The remaining groups of short stature (skeletal disorders, genetic short stature, chronic disease, malnutrition) usually are rather obvious at the time of presentation. The patients with Turner's syndrome were a notable exception to this statement. In this study, the only consistent feature of girls with Turner's syndrome was the short stature. In fact, the classical textbook descriptions of Turner's syndrome may be exceptional. The features of Turner's syndrome can be subtle and escape the notice of the incidental observer. For this reason, we recommend chromosomal analysis on any girl with proven short stature. This will allow diagnosis of this condition prior to recognition of the primary amenorrhea resulting from the gonadal dysgenesis.

The preceding discussion does not address the problem of a physician confronted with a child for the initial evaluation of short stature. After a complete physical examination, we routinely obtain a sedimentation rate to rule out occult chronic inflammatory disease (especially Crohn's disease), a true thyroxine (especially in girls and in those children with palpable thyroid abnormalities), a chromosomal analysis or buccal smear on short females, and a bone age. Next, the parents are asked to search for any height measurements that might provide meaningful longitudinal data. In the absence of any abnormality, the patient is requested to return in six months for another height measurement to ascertain if the growth rate is above the minimum of 5 cm/yr.

By applying the above mentioned guidelines to the evaluation of the child with short stature or growth failure, an appropriate understanding and diagnosis of the majority of these children can be made. In addition, those children with short stature and growth failure who are in need of further evaluation and treatment can be identified.

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PLEASE NOTE

This is a reminder that the first House of Delegates meeting will be on Sunday, May 7, 1978, at the Downtown Ramada Inn, Topeka, beginning at 9:00 A.M. All resolutions will be introduced at that time.

All delegates are urged to participate in the deliberations of Society business.

CLAIR C. CONARD, M.D., *Speaker*

Headache Clinic Functions

The Search for Some Answers

DEWEY K. ZIEGLER, M.D., ***Kansas City, Kansas***

The experience of a headache clinic at KUMC in dealing with nearly 1000 headache patients is described. The evaluation of the patient—through prepared questionnaires, direct confrontation, and diagnostic studies—is utilized to differentiate the several common causes of headache. Management of the patient with headaches with emphasis on patient education is stressed.

VAST SUMS of money spent in the advertising of headache remedies are the most convincing evidence of the high prevalence of this symptom in American society. This prevalence has been confirmed by surveys of symptoms bringing patients to physicians' offices—headache is near the top of the list. Although not a lethal disorder, it is the cause of frequent lost workdays, of missed recreational opportunities and, in many, of bedridden days with severe pain.

Several years ago, the staff of the Neurology Department at Kansas University Medical Center decided to offer a weekly specialty clinic devoted to the study and therapy of patients with this complaint. Nearly 1000 new patients have been examined since that time, and most of these have been seen for one or more follow-up visits. The following information is drawn from that experience. It is intended to aid physicians in all specialties who encounter patients with this complaint and treat the majority without the benefit of consultation.

We have found it advantageous to inform all patients referred to the clinic that accurate information about the frequency and severity of their headache is essential; they are sent, with the appointment time, a calendar on which they are asked to record daily presence of any headache, whether the headache is mild, moderate, severe or disabling, and whether it is accompanied by other symptoms (nausea, scotomata). On arrival at the clinic they are given a long questionnaire containing an exhaustive (and on occasion exhausting) list of questions relating to their life history and the various types of headache (frequency, duration), their perception of precipitating events, accompanying symptoms, and past experience with medication. The questionnaire also contains questions relating to medical history, family history of headache, and psychological symptoms. Data is subsequently placed on data cards and taped for later statistical research. This form, along with the calendar brought by the patient, serves as an excellent introductive summary for the physician to the patient's problem. The physician then obtains a "live" history (often using as clues responses on the questionnaire), performs a general physical and neurological examination and, during both, attempts to evaluate the psychological status of the patient—particularly as is relevant to this complaint. X-ray of the skull is routine, although yield of relevant abnormal findings is extremely low. While computerized tomography may be a far more revealing test, it will be negative in the majority of patients, and we do not feel justified in escalating medical care costs by making this test routine in the vast population of headache sufferers. More rational would be some innovative way of obtaining insight into the comparative importance of the role of emotional factors in the genesis of the headache in each individual patient, and our staff psychologist is now exploring this field.

What are the most frequent next steps? For an occasional patient, hospitalization is recommended for one of two reasons.

In some individuals the headache gives clues to the physician that it is symptomatic of some intracranial or generalized disease and demands a more extensive evaluation, including spinal fluid examination. One can only say that this decision derives from the clinical experience of the neurologist, which tells him intuitively that certain features of this patient's headache, *e.g.* its novelty, severity or accompanying neurological phenomena, suggest known disease processes. This decision is made uncommonly. In the small proportion of cases where headache seems probably related to otorhino-

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laryngological disease, ophthalmic dysfunction or malignant hypertension, appropriate specialist consultation is sought and judgment deferred. In occasional patients it is so clear that a major psychiatric disorder (depression, schizophrenia) is present that immediate psychiatric treatment, often hospitalization, is indicated.

For the other patients, by far a larger number, the next step is a careful statement to the patient that the results of examinations have been negative, that this fact plus the history leads the physician to the conclusion that the patient does not have any life-threatening disease such as tumor or infection of the nervous system. It is sometimes difficult for physicians to realize that a significant percentage of patients have the unspoken fear that they are harboring a brain tumor, or an incipient stroke—convictions often deriving from family history—and that simple reassurance in these patients is curative.

The statement, "You do not have serious disease," is avoided; for any patient suffering from severe recurrent headache will be affronted by such a demeaning of his symptom.

There follows a small lecture on headache with the physician serving as a guide to the patient in the wilderness of uncertainties and controversies incidental to this subject. We have found it helpful to tell patients that the tendency toward headache should not be thought of as analogous to appendicitis or pneumonia. The susceptibility is rarely cured and is often recurrent, but symptoms can be helped and the "illness" is not dangerous. The patients are also told that while the terms "migraine" and "tension headache" are in common use, there are no truly objective ways of making these diagnoses, and the two often overlap. It is also our practice to tell patients that psychological factors are probably important in varying degrees in many patients, and that in any individual case the emotional factor can be determined only with difficulty. At this point the patient is asked if, in his/her opinion, emotional stress may be causative. It is helpful to ask, as an aid to the patient, if he/she recalls whether headaches are clearly related only to time of stress or immediately post-stress, or whether, conversely, they occur during holidays and periods of amusement.

By now the physician has decided whether the weight of evidence tends more to tension headaches or migraine in this individual patient, and this determination is related to the patient.

A combination of reassurance, exploration of possible alleviation of environmental stresses, and pharmacotherapy are employed for tension headaches. The prescription of mild tranquilizers (diazepam) is always accompanied by advice that the major purpose is diagnostic (*i.e.* to see if the symptom is relieved) and the drug should not be used in excess of the prescribed dose or with alcohol, and not for the long term. A decision on psychotherapy is best left to the patient, and should be determined on the basis of emotional distress rather than headache.

When the patient's symptoms are predominantly migrainous with some or all of the following characteristics—long headache-free intervals, presence of vomiting, visual and other neurological phenomena—this syndrome also demands explanation to the patient. The difference between therapy of the acute attack and prophylaxis is explained. The vasoconstrictive action and the absence of analgesic action of ergotamine are clarified. The difficult matter of analgesics for the acute attack is also discussed, with strong encouragement for reliance on sedatives rather than opiates during these difficult periods, and discouragement of parenteral medication.

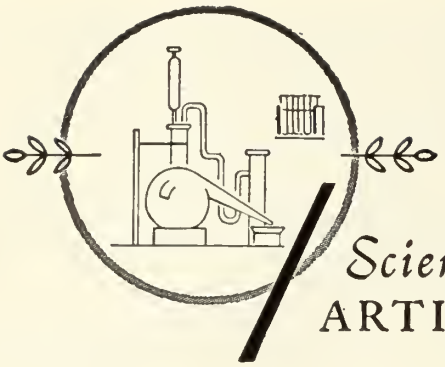
If migraine attacks are frequent (as they usually are in the referred patients) a choice is made between the three agents currently used for prophylaxis: methysergide, amitriptyline, propranolol—and appropriate instructions are given.

Occasionally seen is a third, and more uncommon kind of episodic headache, variously named "cluster headache" or "migrainous neuralgia." In the opinion of some, this syndrome is a variant of migraine; others believed it to be a syndrome *sui generis*. The pain is extremely severe although brief. Numerous prophylactic therapies are being evaluated.

The prevalence of headache dictates that primary physicians will be treating most of these patients. The major service task is one of education—teaching patients the facts about headache, and the uses and limitation of medicine, initiating the disruption of destructive self-medication patterns, and offering the simple benefits of reassurance and interest. Occasional patients can be helped to modify life styles—the driven person to make recreation a part of life, the exceptionally delicate to take up some athletics. All patients are advised of the extraordinary importance of maintaining continued contact with a trusted family physician who will soon know his patient's reaction to drugs, personality, and life situation.

Much has recently been written about biofeedback and acupuncture treatment for headache. Whether

(Continued on page 114)



Scientific
ARTICLES

Embolic Occlusion

Transcatheter Arterial Embolization of Renal Tumors

KYO RAK LEE, M.D.,* JESUS ZORNOZA, M.D.,*
LAWRENCE R. BIGONGIARI, M.D.,* JOHN W. WEIGEL, M.D.†
and BARRY S. FARBER, M.D.,† *Kansas City, Kansas*

Experiences with this non-surgical procedure in two patients with malignant renal tumor are reported. The rationale, technique, and results are discussed.

SELECTIVE TRANSCATHETER arterial embolization has been increasingly used as an alternative procedure to surgical intervention for various clinical conditions including gastrointestinal bleeding; traumatic renal, splenic and pelvic bleeding; and epistaxis and arteriovenous malformations of the central nervous system.[1–3] This has been recently utilized for the preoperative or palliative management of renal carcinomas.[4–8]

Presented here is our experience with transcatheter arterial embolization of renal tumors. The rationale of arterial embolization in the management of malignant renal tumors will also be discussed.

Case One

A 71-year-old white female was admitted to KUMC suffering recurrent hematuria of three months duration and weight loss of 27.3 kg (60 lbs).

Physical examination was unremarkable. Laboratory study revealed moderate anemia with hemoglobin 10.5 mg/100 ml; hematocrit reading, 22%; urine protein, 3+; white blood cell count (WBC), 1-3 per high power field (HPF); red blood cell count (RBC), packed; hemoglobin, 4+ by dipstick; blood urea nitrogen (BUN), 13 mg/100 ml; creatinine, 1.2 mg/100 ml.

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An intravenous pyelogram showed a non-functioning right kidney. The selective renal arteriogram demonstrated a large vascular tumor involving the right kidney *(Figure 1A)*. The left kidney was normal. The diagnosis was hypernephroma of the right kidney.

On the fourth hospital day, the patient developed massive hematuria and her hemoglobin dropped to 7.9 mg/100 ml. She developed progressive renal failure. She was treated with blood transfusion and conservative therapy, and improved over the following two weeks. Since she was considered a poor candidate for nephrectomy, tumor embolization was elected for palliative management to control her continuous hematuria. The tumor was partially embolized by injecting Gelfoam selectively into the right renal artery through a catheter *(Figure 1B)*. She did well after embolization; the hematuria subsided immediately. The patient was discharged in satisfactory condition one week later.

Case Two

A 56-year-old white male was referred to KUMC

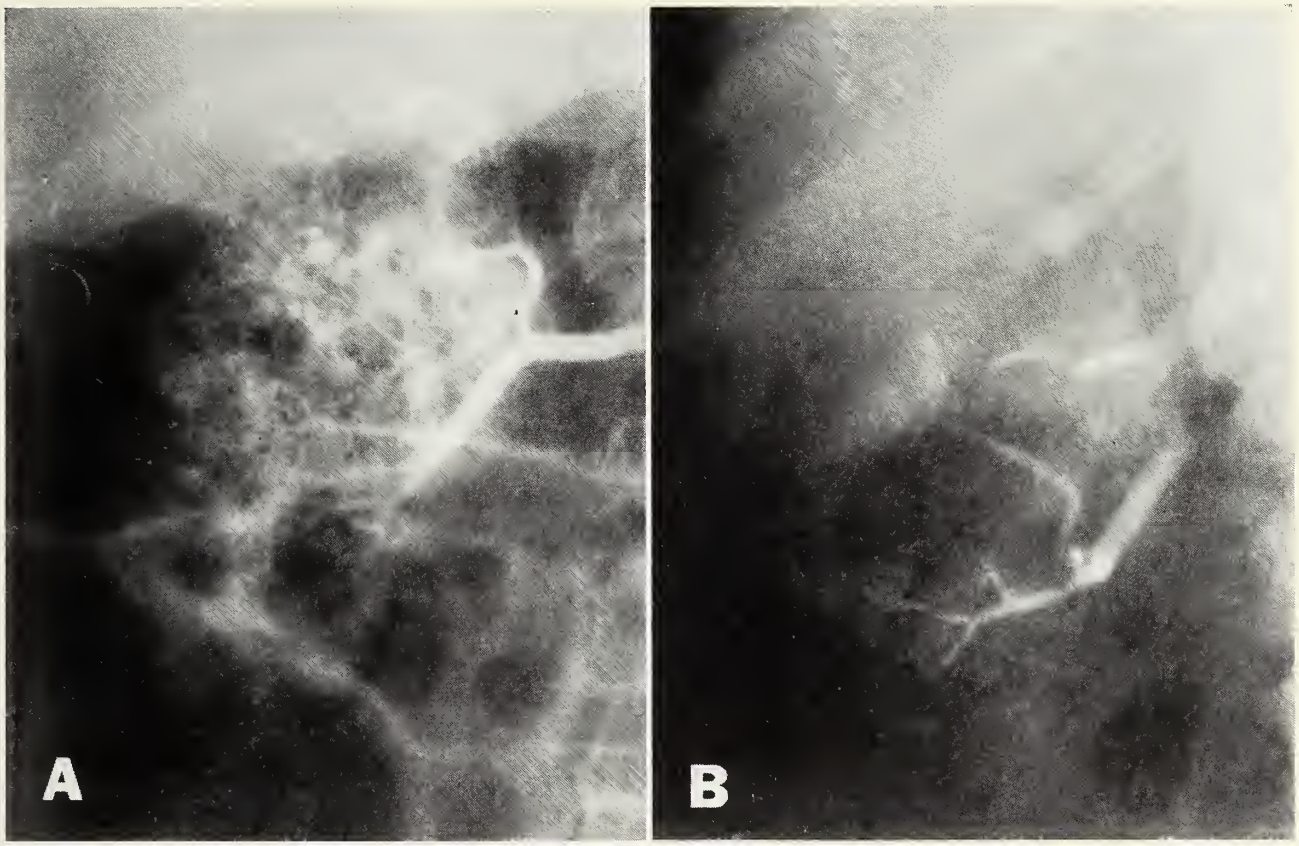


Figure 1. Case one. A selective right renal arteriogram (A) demonstrates a large hypernephroma which was partially embolized by injection of Gelfoam (B).

suffering an abdominal mass that had been detected one month prior to admission. An intravenous pyelogram and renal arteriogram performed at a local hospital showed a large vascular mass in the right kidney. The patient denied hematuria or other urinary tract symptoms.

Physical examination revealed a well developed and nourished male in no acute distress. A large, firm, nontender mass was palpable on the right side of the abdomen. No bruits were detected. The findings of routine laboratory studies were within normal limits.

The results of a routine chest roentgenogram and whole lung tomograms were normal. Excretory urography showed a large tumor involving the right kidney. The tumor contained curvi-linear calcifications. The selective renal arteriogram demonstrated a huge hypervascular tumor measuring 15 × 20 cm and occupying almost the entire right side of the abdomen *(Figure 2A)*. The renal vein was completely closed. An inferior vena cavogram showed a large intracaval thrombus extending from the level of T-12 into a portion of the right atrium. Transcatheter embolization of the right renal artery was performed three days later in order to reduce the tumor bulk for surgery. The right main renal artery was completely embolized by injecting six stainless steel coils and multiple pieces of Gelfoam *(Figure 2B)*. The patient did well after the procedure except for minimal flank pain.

The patient underwent surgery six days after embolization. A large infarcted tumor was found in the right kidney. The tumor was substantially smaller than that seen on the initial arteriogram. After careful removal of thrombus from the inferior vena cava and right atrium by cavotomy and right atrial exploration, the entire tumor and kidney were removed. No local metastasis was present.

The gross specimen measured 23 × 15 × 11 cm and weighed 2 kg 110 gm (4 lb 7 oz). The kidney was almost totally replaced by partially necrotic neoplasm. The tumor was lobulated, very soft, and hemorrhagic. The histologic diagnosis was low grade leiomyosarcoma of the right kidney. After an uneventful postoperative course, the patient was discharged in good condition. The one-month follow-up study revealed that the patient was doing well and had gained 4.5 kg (10 lb).

Discussion

There are three clinical situations for which embolic

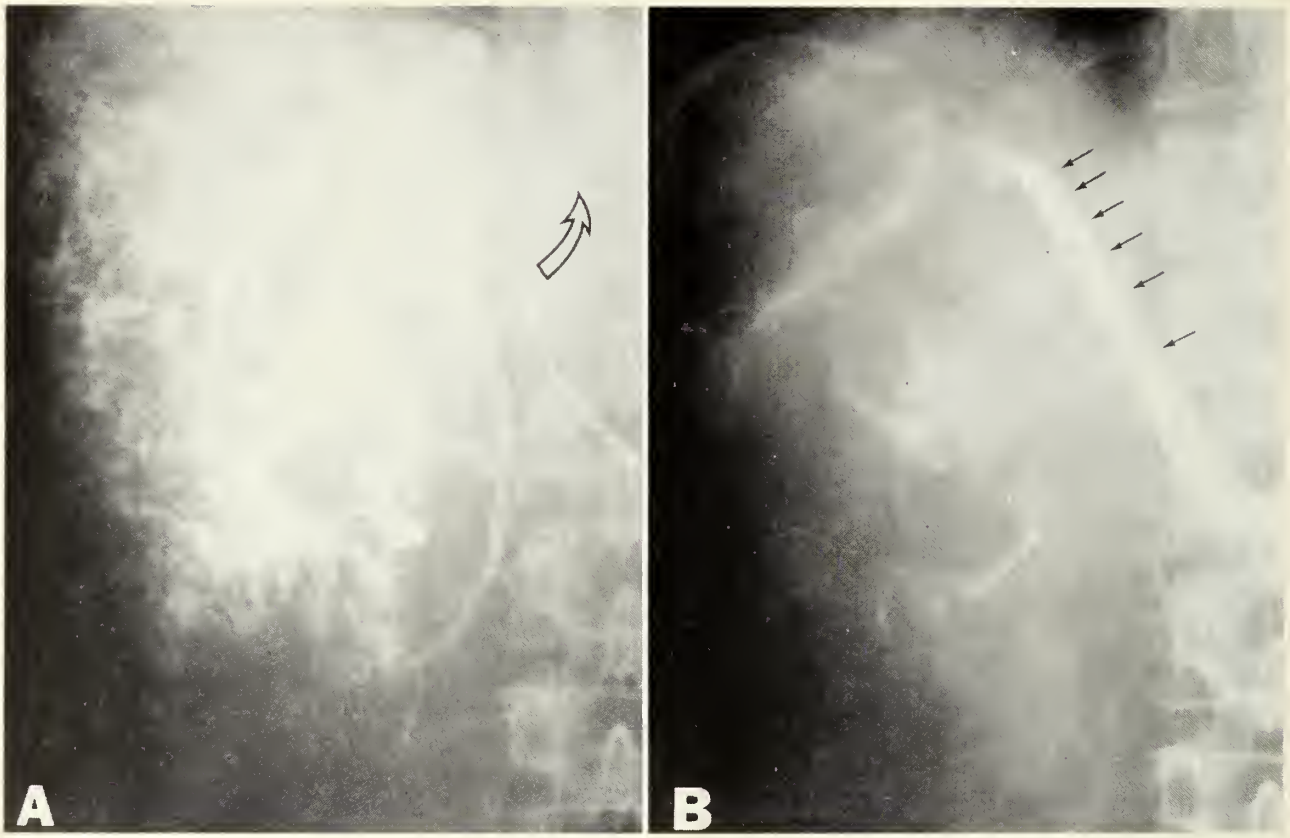


Figure 2. Case two. (A) The selective renal arteriogram demonstrates a huge vascular tumor of right kidney occupying most of the right abdomen. Notice the large arterio-venous shunt (arrow). (B) The postembolization arteriogram shows complete occlusion of the right renal artery with six stainless steel coils (arrows).

occlusion of malignant renal tumors has been utilized: (1) reduction of the tumor mass preoperatively to facilitate the removal of huge vascular tumors (Case 2); (2) palliation of hematuria or pain (Case 1); and (3) reduction of total tumor mass in inoperable cases, alone or in combination with chemotherapy or radiation therapy.[4, 6, 7]

Tumor embolization is technically simple and relatively safe. A size 6 or 7F angiographic catheter is selectively placed in the distal renal artery to be embolized with the usual percutaneous approach. Embolic materials are injected through the catheter under fluoroscopic control. The embolic materials being used include autologous tissue and clot, Gelfoam, metallic and silastic spheres, various silicone preparations, isobutyl-2-cyanoacrylate, and radioactive particles.[6, 9, 10] Stainless steel coils have been recently developed.[11] We prefer a combination of Gelfoam and stainless steel coils for complete embolization.

Surgical removal of the kidney and tumor after embolization is simpler because the tumor is distinctly smaller and less vascular. The risk of tumor embolization through the collapsed venous bed is substantially lessened.

Histological examination in experimental animals showed large areas of necrosis in the renal parenchyma one week after embolization.[4, 7] Areas of living tissue were found between the infarcted areas throughout the cortex and medulla. The kidneys became shrunken and hyalinized 6-10 weeks after embolization. In human cases, a marked degree of necrosis of the tumor and the adjacent renal tissues was seen in the surgical specimen. Total necrosis of the kidney following embolization has not been found either in the experimental animals or in any series of human cases. This is thought to be due to collaterals via the capsular, pelvic, and ureteric vessels.

The number of cases in the present series is small, but long-term results following embolization for inoperable patients are available in the literature. Five of the six patients in Goldstein's series[6] were alive and doing well in follow-up evaluations ten months after embolization. One of them was embolized for relief of flank pain. The tumor was no longer visualized on in-

travenous pyelography and the symptoms had completely disappeared. In Almgård's,[4] eight patients were not followed with surgery after embolization. Six of them had widespread metastasis and the other two were in poor general condition. Five of these patients died of their primary disease, 1, 2, 5, 6, and 12 months after embolization. The remaining three were alive 5, 11, and 26 months later with unchanged metastasis in two and with slight progress in one case.

A transient rise of temperature or a mild to moderate degree of pain and tenderness over the kidney are common symptoms during the first few days after embolization.[4] These symptoms disappear gradually; a few patients may require analgesics. No serious complication occurred in our series or in those of others. No permanent side effects, such as sustained hypertension or hematuria, have been reported.[4, 6]

Summary

Experiences with transcatheter arterial embolization in two patients with malignant renal tumor were reported. Preoperative embolization facilitated surgery upon a huge vascular tumor in one patient, and a therapeutic embolization for continuous hematuria was achieved in another with an inoperable renal cell carcinoma. The rationale, technique, and results of transcatheter embolization of malignant renal tumors were discussed.

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We would like to take this opportunity to thank all of you for your support these past three years. We will be stepping aside and making room for a new chairman, but ask that you remain faithful to our cause and be as generous in the future as you have been to us in the past.

Thanks again,
Caryl *Mary Ruth*

Percutaneous Nephrostomy

A Non-operative Temporary or Long-Term Urinary Diversion Procedure

LAWRENCE R. BIGONGIARI, M.D.;* KYO RAK LEE, M.D.;*
JOHN WEIGEL, M.D.† *and* JOHN FORET, M.D.,† *Kansas City, Kansas*

Cases utilizing this non-surgical approach are outlined and various procedures involved are discussed.

PERCUTANEOUS NEPHROSTOMY provides drainage of an obstructed urinary system with minimal patient discomfort. Although the percutaneous approach was described more than 20 years ago,[1] and numerous articles have accumulated,[2–17] the procedure has not gained wide acceptance. It is particularly helpful as a temporizing measure in the patient who is a poor surgical risk because of infection, uremia, or other factors. It can also be used to achieve long-term or permanent diversion in selected patients. The following cases are representative of our experience with this procedure at the University of Kansas Medical Center.

Case One

A 66-year-old white female who had had a pelvic exenteration for cancer of the cervix with colostomy and ureterocolostomy five years previously was brought to the emergency room by relatives. She had become progressively disoriented and confused over the previous two days. She also experienced nausea, vomiting, and anorexia. The laboratory tests produced the following results: BUN, 86 mg/dl; serum creatinine, 5.3 mg/dl and CO, 9 mEq/l; WBC, 9.4 × 10.3. The patient was afebrile. After hydration and correction of metabolic acidosis, she remained obtunded. Excretory urography three days after admission *(Figure 1)* showed bilateral hydronephrosis with filling defect on the right side, suggesting pyonephrosis. Bilateral percutaneous nephrostomies were performed *(Figure 2)*, with placement of a 6F catheter in the left kidney and direct insertion of a 12F Ingram Trochar catheter in the right kidney. Clear urine was obtained from the left kidney. Frank, foul smelling pus was obtained from the right kidney, which subsequently cultured Klebsiella, Enterobacter, and E. Coli. The ward staff noted that the patient was more alert on her return to the floor. She was fully oriented and alert 48 hours later. Three days after the procedure, BUN was 29 mg/dl and serum creatinine 2.3 mg/dl. A low grade fever was resolved with antibiotics by the fourth day. One week after percutaneous nephrostomy a transverse colon conduit was created. At the time of surgery BUN was 17 mg/dl and creatinine 2.2 mg/dl. The nephrostomy tubes



Figure 1. Case I: Urogram demonstrating marked bilateral hydronephrosis with filling defect on the right.

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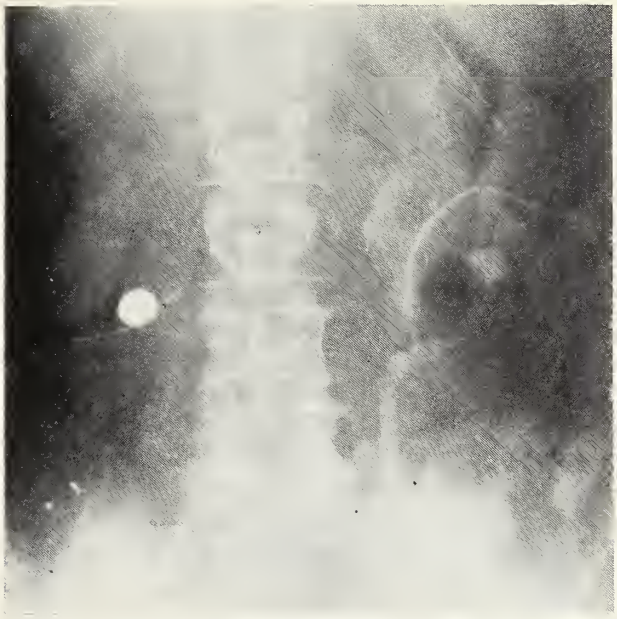


Figure 2. Bilateral percutaneous nephrostomies using direct insertion of a 12F Ingram Trochar catheter on the right and a straight 6F angiographic catheter on the left with its tip in the proximal ureter.

were removed one week after surgery and the patient was later discharged in good condition.

Case Two

A 57-year-old obese white female presented with bilateral pedal edema and an enlarged left supraclavicular node. She had received intracavitary radiation therapy two years previously for squamous cell carcinoma of the uterine cervix. Upon admission, BUN was 35 mg/dl and creatinine 2.2 mg/dl. Excretory urography *(Figure 3)* demonstrated a calcified gallstone, a small atrophic right kidney, and left hydronephrosis with proximal hydroureter. Retrograde pyelography showed a 3-cm extrinsic narrowing of the distal left ureter and a 1-cm extrinsic narrowing of the distal right ureter. A ureteral catheter could not be passed through the strictured left ureter.

A left percutaneous nephrostomy via a lower pole calyx, using a 6F angiographic catheter, provided decompression and good urine flow. The patient was discharged the next day to receive radiation therapy to the recurrent pelvic neoplasm on an outpatient basis. Two weeks later, the tract was enlarged and a 10F catheter was positioned in the proximal left ureter. Six weeks after that, it was apparent that more permanent drainage was needed and a 12F Ingram catheter was substituted. Since its balloon could not be inflated without blocking the lower pole infundibulum, the catheter was only sutured to the skin. About four weeks later, the patient inadvertently pulled out the tube by stepping on the collection tube. She did not seek medical attention before the tract closed because she was passing urine via the urethra. During a subsequent admission, excretory urography *(Figure 4)* showed that the left hydronephrosis was considerably reduced from the earlier study. A left ureteral catheter was then introduced, and the patient was subsequently discharged from the hospital in satisfactory condition.

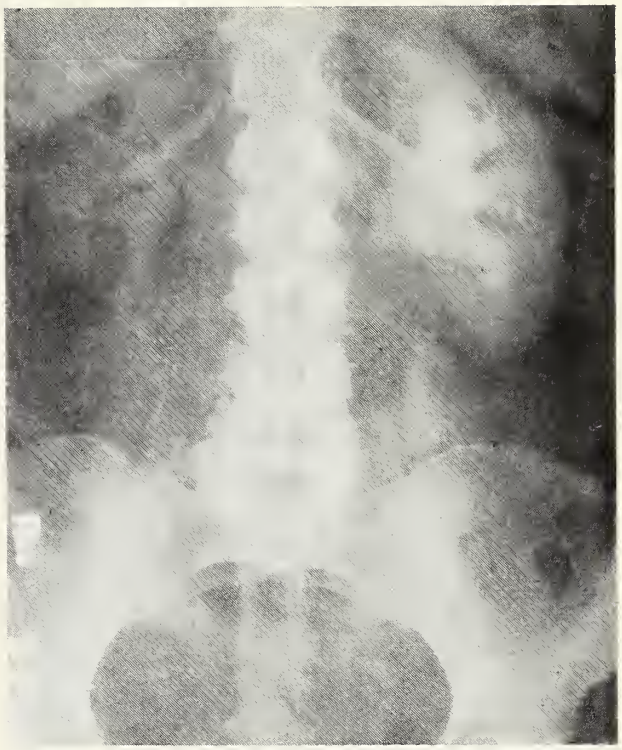


Figure 3. Case II: Initial urogram demonstrating a small right kidney and left hydronephrosis with obstruction of the lower ureter.

Discussion

Our technique is individualized to the particular patient and the clinical circumstances to treat even a mild or moderately dilated urinary tract. We prefer an approach lateral to the paraspinous muscles rather than the shorter posterior approach. The patient is positioned prone oblique so that a direct vertical puncture will enter the collecting system. The more lateral position of the tube affords the patient more comfort in the supine and sitting positions, particularly when bilateral procedures are performed. We believe that the lateral approach through renal parenchyma provides better fixation of the catheter, less bleeding and leakage, and even actual tamponade compared to the direct posterior approach into the renal pelvis. Ideally, the catheter transverses the bloodless plane between the anterior and posterior renal lobes.

The renal pelvis is first visualized by ultrasound, ex-



Figure 4. Case II: Follow-up urogram after percutaneous nephrostomy and radiation therapy demonstrates marked resolution of the hydronephrosis. A left ureteral catheter was subsequently placed.

cretory urography or preliminary antegrade pyelography with a fine gauge needle. In massively dilated systems, overhead films and fluoroscopic control will provide adequate localization. A fine needle may be used for depth sounding as well as initial antegrade pyelography. An 18 gauge Teflon sheathed needle, such as is used for translumbar aortography or spleenoportography, can then be directed under local anesthesia below the 12th rib into the renal pelvis or a dilated calyx. The sheath is exchanged over a J tip, movable core guide wire, the tract dilated, and a preshaped 6 to 7F angiographic catheter with at least 10 side holes inserted. This catheter can be positioned in the proximal ureter, upper infundibulum, or coiled in the pelvis depending on the particular patient's anatomy. With obstruction relieved, the system can be expected to shrink. Positioning in the proximal ureter allows a generous length of catheter to be inserted in the system as precaution against displacement by respiration or patient movement. A 6 or 7F catheter will provide adequate urine drainage; but if blood clot, pus or debris is present, the tract should immediately be dilated to 10-12F for adequate drainage. A massively dilated collecting system, particularly in an obtunded patient, will allow safe direct insertion of any large-bore, commercially available suprapubic drainage system. If long-term diversion is needed, the tract should be made large enough to accommodate a standard nephrostomy appliance, such as an 8-10F Foley or mushroom catheter.

Whatever catheter is used, it must be securely fixed to the skin with suture material until a tract forms. A granulated tract will form in 10-14 days. If the catheter is removed sooner, another puncture will be necessary. The catheter is padded by cut gauze sponges, to prevent kinkage at the skin, and is connected to a drainage bag.

Transient hematuria is frequent, but we have encountered no difficulty with bleeding. Antibiotic coverage should be provided against risk of sepsis. In their series, Almgard and Fernstrom[2] reported one instance of perforation of a catheter into the adjacent ascending colon, apparently spontaneously, one week after insertion. No further complications occurred after the catheter was removed. Use of a soft catheter should preclude this complication. Burnett *et al.*[6] experienced one case of perforation of the renal pelvis into the retroperitoneum during insertion. The catheter tip was left outside the pelvis for drainage, then pulled back 10 days later. The patient's course was stable throughout. We have encountered no serious complication in our use of this technique, and are aware of no reported mortality in the literature.

Percutaneous nephrostomy is quick and safe in skilled hands. Mastery of the techniques of cyst puncture and angiography are basic to this procedure. The procedure should be used when surgical intervention either is not feasible or not indicated. Percutaneous nephrostomy can be a temporizing measure until the patient's condition allows definitive surgical diversion or correction. It can provide palliation in the terminally ill without surgery or prolonged hospitalization. Informative, high quality antegrade pyelography can be readily obtained via the indwelling catheter, and therapeutic efficacy can be easily assessed. If infection is present, specimen for culture and sensitivity can be obtained directly from the kidney, while making available a route for irrigation.

Conclusion

Percutaneous nephrostomy can provide urinary diversion in most patients without complication or prolonged hospitalization. It allows the surgeon to perform definitive correction of obstruction under more favorable circumstances than exist in cases of patients with uremia or pyonephrosis. It provides nonoperative pal-

(Continued on page 132)

Adjuvant Immunotherapy

Treatment of High Risk Patients With Malignant Melanoma

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An extensive experience with active adjuvant immunotherapy in 80 patients is reviewed. The results confirm the reports of other authors and suggest that clinical investigations in this area should be continued, allowing for thorough evaluations of the many patient and treatment variables.

SURGICAL MANAGEMENT of the patient with malignant melanoma has been most effective in patients with minimal local invasion and absence of regional nodal metastases. The accepted surgical therapy remains wide local excision of the primary lesion, with prophylactic lymphadenectomy where applicable. However, recent data from an evaluation of prophylactic lymphadenectomy by the World Health Organization Melanoma Group seriously questions its efficacy in improving survival.[1]

Treatment failures in patients with proven nodal metastases following lymphadenectomy approach 80 per cent,[2] and are usually secondary to distant metastases, suggesting a similarity to surgical failures in breast cancer. Since more than 75 per cent of melanoma metastases become apparent within two years, as is true in breast carcinoma, microscopic subclinical disease is probably present at the time of the initial surgical procedure.[3]

Although the presence of positive regional lymph nodes is the most effective indicator of the "microdissemination" of melanoma, the "microstaging"[4, 5] may influence the pattern of metastases. Clark and other investigators have described decreased survival with melanomas extending below the reticular dermis (Clark's levels IV and V).[6–8] Whether this poor survival is related to nodal metastases is not well defined but, when considering a primary involvement in a site of ambiguous lymphatic drainage, the level of the lesion must be considered the major determinant of survival. Likewise, Breslow[9, 10] has described decreased survival with primary melanomas of greater than 1.5mm in depth as measured by an ocular micrometer. Again, the incidence of nodal involvement associated with the depth of invasion may influence this decrease in survival, but the depth of invasion should be considered the major determinant of longevity in appropriate situations.

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Consequently, the patients with melanoma at high risk for the development of recurrent disease include not only those with regional nodal metastases, but also patients with primary melanomas with the above mentioned microstaging characteristics—that is, Clark's level IV and V—and depth of invasion greater than 1.5mm.

In an attempt to improve the results of therapy in high risk patients with malignant melanoma, systemic adjuvant antitumor therapy was undertaken by the Surgical Oncology Group at the University of Kansas. The initial report of this experience by Jewell *et al.*[10] described the results of adjuvant immunotherapy utilizing an acellular allogenic melanoma preparation in 44 high risk patients. Thomas *et al.* have demonstrated the effectiveness of this allogenic tumor preparation in preventing tumor growth, in an immunoprophylactic model using a methylcholanthrene-induced subcutaneous tumor in C57-6J mice.

The following report updates our experience with adjuvant immunotherapy in high risk melanoma patients.

Patients and Methods

Eighty patients participated in this study from 1972-1977. All patients were evaluated by history, physical examinations, chest x-ray, routine laboratory tests, and liver, brain and bone scans. The studies were performed to exclude the possibility of distant metastases. Surgical management included a wide local excision

TABLE I

CUMULATIVE SURVIVAL RATES FOR STAGE II MELANOMA PATIENTS

1	2	3	4	5	6	7	8	9
0-6	43	2	0	2	42	.048	.952	.952
7-12	39	7	0	6	36	.194	.806	.767
13-18	26	0	0	4	24	0	1	.767
19-24	22	3	0	2	21	.143	.857	.657
25-30	17	0	0	3	15.5	0	1	.657
31-36	14	2	0	1	13.5	.148	.852	.560
37-42	11	0	0	1	10.5	0	1	.560
43-48	10	1	0	2	9	.111	.889	.498
49-54	7	0	0	2	6	0	1	.498
55-60	5	0	0	2	4	0	1	.498
61-66	3	0	0	1	2.5	0	1	.498
67-72	2	0	0	2	1	0	1	.498

1. Months after treatment initiated
2. Number alive at beginning of interval
3. Number dying during interval
4. Number lost to follow-up during interval
5. Withdrawn alive during interval
6. Effective number exposed to risk of dying
7. Proportion dying
8. Proportion surviving
9. Cumulative survival (× 100)

and regional node dissection where applicable. Several patients were referred after appropriate surgical therapy elsewhere.

Forty-three of the 80 patients were considered high risk because of the presence of nodal metastases by microscopic evaluation, and 37 because of the location of the lesion, depth of invasion, or level of invasion.

Forty-two per cent of all primary lesions were located on the trunk, and six patients presented with nodal metastases without evidence of a primary lesion.

Four to six weeks after operation, patients were started on an adjuvant course of the allogenic tumor preparation. Subcutaneous injections were given weekly for two months followed by booster injections at three-month intervals. Exchange of white blood cells has been discontinued since the initial report of Jewel *et al.* No adverse reactions to the tumor preparation were identified.

The preparation of the tumor extract has been described previously.[10] Briefly, using sterile technique, a 20 per cent-weight/volume suspension of melanoma tumor was rendered acellular by rapid freezing and thawing. The homogenate was then centrifuged four times, the final centrifugation being 102,000 × g for 30 minutes. The final supernatant was concentrated 10:1 by Diaflo vacuum filtration. The final tumor equivalent was 2mg of tumor/ml/extract.

All patients were reevaluated as determined by the injection schedule. Repeat chest x-ray and radioisotope scans were obtained at a minimum interval of three-six months. Only two patients were lost to follow-up observation.

Time to recurrence was based upon the time from initiation of immunotherapy to the first clinical evidence of recurrence. Every effort was made to obtain pathologic confirmation of recurrent disease where possible. Statistical analysis of the study was performed by the Life Table method as described by Cutler and Ederer.[11] Cumulative survival and cumulative disease-free rates were calculated.

Results

Survival and recurrence data were determined separately for the 43 high-risk patients with nodal involvement, and the 37 high-risk patients with no nodal metastases. The survival and recurrence data for the patients with nodal involvement are shown in *Tables I and II.*

All the patients were found to have clinically positive nodes prior to lymphadenectomy, thus increasing significantly the risk of recurrent disease. In fact, Knutson *et al.* found a cumulative five-year survival rate of 20 per cent in 40 patients with clinically and histologically positive nodes.[3] The survival rate had decreased to 7 per cent at ten years. The five-year survival rate for this group of patients treated with adjuvant immunotherapy is 50 per cent. Additionally, there appears to be little evidence for recurrence after three years, although the majority of patients have been at risk for less than an optimal time period.

TABLE II

CUMULATIVE DISEASE FREE RATES FOR STAGE II MELANOMA

1	2	3	4	5	6	7	8	9
0-6	43	11	0	2	42	.262	.738	.738
7-12	30	5	0	4	28	.178	.822	.607
13-18	21	2	0	3	19.5	.102	.898	.545
19-24	16	4	0	1	15.5	.258	.742	.404
25-30	11	0	0	2	10	0	1	.404
31-36	9	1	0	0	9	.111	.889	.359
37-42	8	0	0	1	7.5	0	1	.359
43-48	7	0	0	2	6	0	1	.359
49-54	5	0	0	1	4.5	0	1	.359
55-60	4	0	0	1	3.5	0	1	.359
61-66	3	0	0	1	2.5	0	1	.359
67-72	2	0	0	2	1	0	1	.359

1. Months after treatment started
2. Without recurrence at beginning of interval
3. Recurrence during interval
4. Lost to follow-up
5. Withdrawn without recurrence
6. Effective number exposed to recurrence
7. Proportion with recurrence
8. Proportion without recurrence
9. Cumulative disease free (× 100)

The survival and recurrence data for the stage I high-risk patients are shown in *Tables III and IV*. No collective criteria are available to evaluate such a heterogeneous group. However, if we assume that these patients may represent those with stage I disease who develop recurrence within three years, then both the three-year survival rate of 96 per cent and disease-free rate of 73 per cent are acceptable figures.

Discussion

It has often been suggested that either specific or nonspecific immunostimulation may be of value to the patient with micrometastases secondary to melanoma. High-risk patients as defined by the above mentioned criteria seem to enjoy a more favorable clinical course when treated with specific active adjuvant immunotherapy.

TABLE III

CUMULATIVE SURVIVAL RATE FOR STAGE I MELANOMA

1	2	3	4	5	6	7	8	9
0-6	37	0	0	7	33.5	0	1	1
7-12	30	1	1	8	25.5	.039	.961	.961
13-18	20	1	1	8	15.5	.064	.936	.899
19-24	10	0	0	5	7.5	0	1	.899
25-30	5	0	0	1	4.5	0	1	.899
31-36	4	0	0	4	2	0	1	.899
37-42								
43-48								
49-54								

1. Months after treatment initiated
2. Number alive at beginning of interval
3. Number dying during interval
4. Number lost to follow-up during interval
5. Withdrawn alive during interval
6. Effective number exposed to risk of dying
7. Proportion dying
8. Proportion surviving
9. Cumulative survival (× 100)

TABLE IV

CUMULATIVE DISEASE FREE RATES FOR STAGE I MELANOMA

1	2	3	4	5	6	7	8	9
0-6	37	1	0	7	33.5	.0299	.970	.97
7-12	29	3	1	8	24.5	.122	.878	.852
13-18	17	0	1	7	13	0	1	.852
19-24	9	1	0	4	7	.143	.857	.730
25-30	4	0	0	1	3.5	0	1	.73
31-36	3	0	0	3	1.5	0	1	.73
37-42								
43-48								
49-54								

1. Months after treatment started
2. Without recurrence at beginning of interval
3. Recurrence during interval
4. Lost to follow-up
5. Withdrawn without recurrence
6. Effective number exposed to recurrence
7. Proportion with recurrence
8. Proportion without recurrence
9. Cumulative disease free (× 100)

Morton *et al.*[2] demonstrated similar results utilizing Bacillus Calmett-Guérin (BCG) in an uncontrolled study of nonspecific adjuvant immunotherapy.

Immunization with tumor-specific antigens is a more effective means of immunotherapy in animal systems than treatment with nonspecific immune stimulants alone, such as BCG. Morton has suggested that immunotherapy combining these two modalities may be more effective than either alone. As recurrences continue to occur with specific adjuvant immune therapy, this approach is an attractive future alternative.

Despite the apparent successes of immunotherapy, the effectiveness, route of administration, type, amount, and duration of therapy remain a matter of conjecture. Studies must be designed to evaluate these variables, especially in the patient with malignant melanoma, because of its well documented response to immunostimulation. The increasing numbers of identifiable patient variables would suggest that these solutions will be defined only through multi-institutional efforts.

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A New Challenge

Measles and Measles Immunization

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Two cases of measles occurring subsequent to vaccination are outlined, and guidelines for immunization are reviewed.

DESPITE EFFECTIVE VACCINES, measles (rubeola) remains a significant contagious illness in children and adolescents. Since the licensure of measles vaccine in 1963, there has been a marked decrease in the overall incidence of measles in the United States. However, the highest nationwide annual incidence since 1971 was reported in 1976, with 39,585 cases.[1] The state of Kansas ranks among the ten states with the highest four-year average incidence rates for the years 1973-76, with an average of 112 reported cases per 100,000 population under 18 years of age. There has been a marked rise in both incidence and total cases among children 10-14 years of age, while the incidence among preschoolers remains highest among all age groups. Krugman[2] estimated that there are approximately five million unimmunized children and adolescents in the United States, and that 35 per cent of 1-4-year-old children are currently unvaccinated. This is a sizable enough population to support the persistence of rubeola.

Important questions concerning the practice of measles immunization have been raised in recent years, specifically the occurrence of measles or atypical measles in the immunized population, and duration of protection afforded by immunization. This paper will present two cases of measles illustrating the varied clinical manifestations, and review current immunization recommendations.

Case One (Vaccine Failure)

A 19-month-old white female was admitted to the University of Kansas Medical Center in April 1977, for a malfunctioning ventriculo-peritoneal shunt. During the hospitalization, an adolescent (Case Two) was admitted with atypical measles to the same ward. A pediatric resident directly involved with the care of both patients subsequently developed measles documented with a significant rise in complement fixation antibody titers to measles virus. Some two days after discharge from the hospital, our patient developed fever, cough, coryza, and conjunctivitis. She was seen at KUMC on the fourth day post dismissal when a rash appeared. A review of her immunization history revealed that Schwartz vaccine was administered at KUMC at the age of 12 months. On physical examination, the temperature was 39.7 C; respiratory rate, 40/min; and heart rate, 130 beats/min. There was a confluent erythematous rash of maculopapular character over the face, neck, and upper arms. The rash was scattered over the lower trunk, and a few macules were noted on the lower extremities. No Koplik's spots were observed. A clinical diagnosis of rubeola was made. The clinical course was uncomplicated. Acute measles complement fixation titer was less than 1 : 8. A convalescent titer drawn one month later was 1 : 64. A throat swab for virus was negative.

Case Two (Atypical Measles)

A 13-year-old white female was in relatively good health until one week prior to admission, when she first developed cough and fever (39.8 C). The following day, a maculopapular vesicular rash was noted on the palms, hands and feet, which spread over the extremities to involve the trunk within a 48 hour period. The patient was transferred to KUMC because of persistently high temperature, tachypnea, and radiographic evidence of progressive pneumonia. Past medical history revealed that the patient had received two killed measles vaccines followed one month later by an attenuated vaccine in 1965. There was an epidemic of rubeola in the community at the time of this admission.

Physical examination of the patient upon admission revealed the following: temperature, 39.8 C; blood pressure, 138/84 mm Hg; respiratory rate 48/min; heart rate, 160 beats/min; mild bilateral conjunctivitis

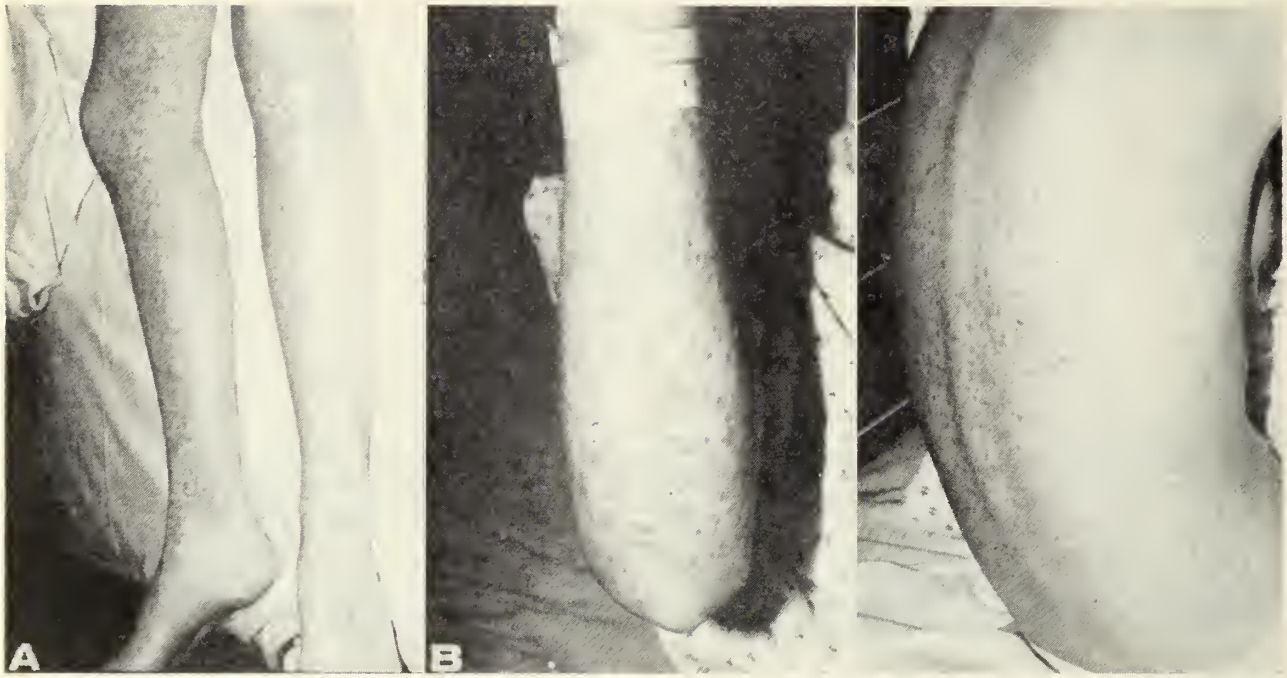


Figure 1. The papular, vesicular hemorrhagic rash of atypical measles (Case Two). Early and extensive involvement of the legs (A) and arms (B), followed by a less severe involvement of back and trunk (C).

and subconjunctival hemorrhages were noted. There was a pruritic maculopapular, hemorrhagic vesicular rash involving primarily the palms, upper and lower extremities, with less involvement of the trunk and back *(Figure 1)*. There were no facial lesions, but there was malar erythema and circumoral pallor. Buccal mucosa was clear. There were moderate costal retractions, and auscultation of the chest revealed bronchial breath sounds on the right, but no evidence of rales. Chest x-ray demonstrated a right-lower lobe pneumonia with interstitial involvement of the left parahilar area *(Figure 2)*. The results of laboratory studies were unremarkable except for a total white blood cell count (WBC) of 23,500/cu mm, with 32% segs, 60% bands, 8% lymphocytes.

Within three days, the patient had defervesced and the respiratory rate was consistently less than 30/min. Over a five-day period, the rash began slowly to fade and became less pruritic. The sub-conjunctival hemorrhages gradually resolved.

Follow-up examination two weeks after discharge showed marked resolution of the rash except for areas of mild discoloration where hemorrhagic vesicular lesions had existed, primarily on the arms and legs. Follow-up chest x-rays showed progressive resolution of the pneumonitis, and by one month, there was no evidence of pulmonary residuum.

Discussion

Clinical presentation

The cases presented here illustrate two examples of the spectrum of illness caused by rubeola virus in immunized children and adolescents: regular measles due to vaccine failure (Case One), and atypical measles (Case Two).

Regular measles: Typical wild type measles infection has an incubation period of 10-11 days. A 3-4-day prodrome period marked by an escalating fever, cough, coryza, and conjunctivitis is followed by the appearance of an erythematous maculopapular rash. Koplik's spots on the buccal mucosa may be seen during the early prodrome period. The rash progresses in a caudad direction, with heaviest distribution over the face, trunk, and upper torso. The fever falls rapidly 1-2 days after the rash appears. Desquamation occurs as the rash recedes on the seventh-ninth day of the illness. The clinical presentation of typical measles occurs when there is no prior host protective, underlying disease.[3]

Typical rubeola infection may be modified when specific or pooled gamma globulin is administered after exposure. This therapy will alter the clinical presentation causing a prolonged incubation period, shorten the prodrome period, diminish the extent of the rash, and decrease the incidence of complications.

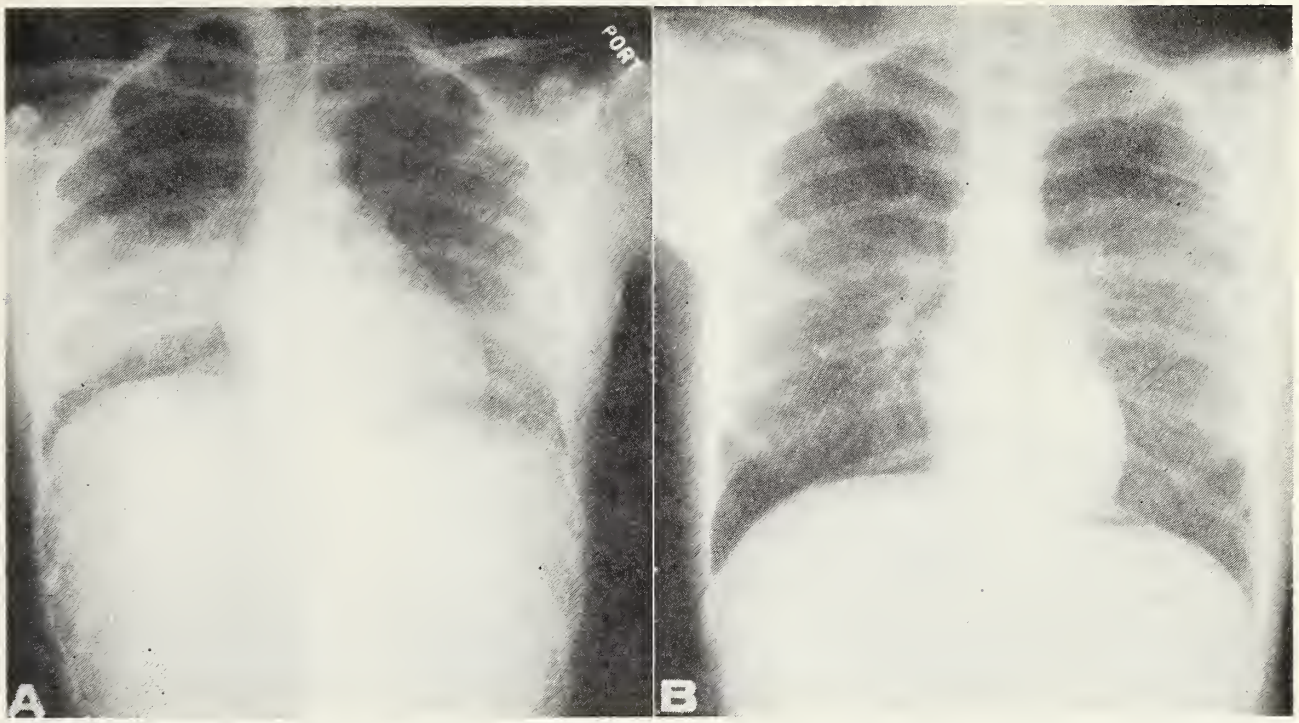


Figure 2. Pneumonia in a child with atypical measles (Case Two). Pulmonary infiltrates were noted on chest film upon admission (A). Follow-up film one month later showed a complete resolution of the pulmonary infiltrates (B).

Atypical measles: In 1965, Rauh and Schmidt[4] described the clinical presentation of measles occurring in patients who had previously been vaccinated with killed measles virus vaccine. These patients present with an abrupt onset of cough, fever, and either abdominal pain or severe headaches. These symptoms precede the onset of rash by 48-72 hours. The rash usually begins on the distal portion of extremities involving the palms, soles, wrists, and ankles (as seen in Case Two). Over the following 3-5 days, further involvement of the extremities, face, and trunk occurs with a cephalad progression. The rash begins as an erythematous maculopapular exanthem, but commonly progresses to papular, vesicular, and petechial lesions. Resolution is usually complete by two weeks. Involved areas are usually edematous and occasionally pruritic.

Pneumonia is a common complication in atypical measles; the pneumonic process may be either a lobular or segmental infiltration with or without hilar adenopathy, and pleural effusions. Residual nodular parenchymal lesions are a common occurrence, persisting for as long as 30 months following resolution of the pneumonia.[5]

Cherry *et al.*[6] described a series of patients who had received attenuated live virus vaccine and subsequently developed a syndrome of atypical measles. In these patients, however, symptoms were less severe and the duration of illness was shorter. Weiner *et al.*[7] recently described a series of adolescents who had seemingly been properly immunized but subsequently developed clinical measles. Case Two exemplifies an adolescent who had supposedly been properly immunized and subsequently developed measles of the atypical variety. The clinical significance of this problem is further demonstrated by the fact that during the Topeka outbreak, 64 of 182 reported cases (35%) occurred in patients with a previous history of vaccination.[1]

Measles vaccine failure

Yeager *et al.*[8] have reported a failure to document protective levels of hemagglutination inhibition antibody titers (less than 1 : 4) in 14.6 per cent of children vaccinated with attenuated vaccine at less than 13 months of age. There was a significant decrease in the percentage (5.2%) of unprotected children when vaccination was performed after 13 months of age. Persistence of maternal antibody beyond 12 months may be responsible for the high percentage of vaccine failure in the early vaccinated group. These data suggest that current immunization practices may be related to the recent increase in the number of cases of measles. Krugman[2] has followed children after immunization for 12 years and has demonstrated that 91 per cent retain protective antibody, while Weiner *et al.*[7] have reported the disturbing observation that measles immunity after vaccination may wane allowing previously immunized

adolescents to develop the disease years after primary vaccination. Other explanations for vaccine failure include: improper storage prior to dispensing and the simultaneous administration of measles immune globulin and vaccine.

Recommendations

The Committee on Infectious Diseases of the American Academy of Pediatrics issued a vaccination policy statement in June 1977.[9] These current recommendations include:

(1) the age of routine vaccination at or after 15 months;

(2) infants at risk of exposure during an outbreak may receive immunization at 6 months of age with revaccination at or after 15 months;

(3) during an outbreak of rubeola, children who were initially vaccinated at or shortly after 12 months of age do not need to be recalled for vaccination;

(4) any child at school entry who does not have a documented history of natural measles or history of live measles vaccination at or after 12 months should receive live measles vaccine;

(5) re-immunization for those children who received live vaccine with immune serum globulin is unnecessary if these were administered at or after 12 months of age;

(6) children who received only inactivated measles vaccine should be given a dose of live measles vaccine and should be warned of the complications of local erythema, heat, tenderness and swelling, as well as systemic complications of fever, malaise, or regional lymphadenopathy;

(7) unvaccinated exposed infants and children should receive 0.25 ml/kg of immune serum globulin intramuscularly (IM) as soon as possible, followed in eight weeks with live virus vaccine unless modified measles develop;

(8) children with lymphoma, leukemia, immunodeficiency disorders, or generalized malignancy should receive 0.5 ml/kg IM (total dose not to exceed 15 ml) immune serum globulin if exposed to measles.

Summary

Measles represents a challenge to clinicians, in terms of both prevention and diagnosis. Immunization remains the only effective means of decreasing the current morbidity and mortality from the disease. With the varied clinical manifestations of rubeola, a high index of suspicion is necessary to ensure diagnosis. Practitioners should be aware of the current vaccine recommendations.

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Headache Clinic Functions

(Continued from page 99)

these new treatments have specificity above and beyond the almost incalculable effect of programmed relaxation and reassurance remains to be seen.

In summary, the headache clinic functions as a source of data for research. Life patterns of headache have not been extensively studied and may yet give insight into treatment results. The uniform data obtained from the headache clinic is being tabulated to this end. It is well known that emotional factors play an important role in some headache attacks. Can we determine the characteristics of the patients in which this role is critical, can we further refine our knowledge of what these emotional factors are, and can we discover in which attacks they seem to be or not to be predominantly involved? Can we determine any other factors which set into play the sequence of events leading to severe headache? Systematic studies from a headache clinic may contribute data toward discovery of answers to these interesting questions.

A Catastrophic Complication

Acute Dissection of an Aortic Aneurysm in a Child With Marfan's Syndrome

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A FATAL dissection of the aorta was suffered recently by a nine-year-old male with Marfan's syndrome. His age and problems in early recognition of the significance of his chest pain prompted a review of the literature for observations regarding this problem in children. The importance of dissection of the aorta in patients with Marfan's syndrome is stressed in a study at Johns Hopkins Hospital.[1] In a series of 157 patients with Marfan's syndrome seen over a 22-year period, approximately 65 per cent of males and 45 per cent of females were alive by age 20. By 30 years of age, this had declined to 35 and 25 per cent respectively. The leading cause of death was dissection of an aneurysm of the aorta along with other forms of aortic involvement. Despite the rather foreshortened survival of patients with Marfan's syndrome, very few instances of children dying from this complication prior to adolescence have been reported. The following is the case report of a nine-year-old boy who died from acute dissection of the aorta, and a review of this occurrence in children with Marfan's syndrome.

Case Report

A 2½-year-old male was first seen at the KUMC Children's Rehabilitation Unit for mental and physical developmental assessment in April 1970. At birth, he had had multiple dislocations involving his hips, knees and metatarsal joints of the great toes, as well as bilateral flexion deformities of his fourth and fifth fingers and calcaneal valgus deformity of his left foot. By the age of four years, he had developed marked kyphoscoliosis, pectus carinatum, radiologic evidence of dilatation of the aorta and a dislocated right lens, leading to the diagnosis of Marfan's syndrome. Pediatric cardiology evaluation demonstrated a systolic thrill in the suprasternal notch, a Grade III+/VI systolic ejection murmur in the aortic area, and a continuous murmur below the left clavicle. The results of the electrocardiogram were normal, but a chest roentgenogram documented a dilated, tortuous aorta.

Cardiac catheterization at the age of six years showed no gradient across the aortic valve. The aortic and left ventricular pressures were 120/76 mmHg and 120/7 mmHg respectively. Other pressures were also normal. Neither aortic nor mitral regurgitation were evident angiographically. A ductus arteriosus was demonstrated by aortic root angiography as well as by an oxygen saturation step-up in the pulmonary artery, yielding a pulmonary to systemic flow ratio of 1.26 to 1.0. Dilation of the sinus of Valsalva was also seen. Surgical obliteration of the ductus arteriosus was not recommended since the ductal shunt was small, his kyphoscoliosis was moderately severe, and the surgical risk would be increased by the friable aortic tissues found in Marfan's syndrome.

A case report emphasizing the ramifications of this rare complication in children is discussed and related literature is explored.

Over the next several years, the patient was repeatedly hospitalized for simple mastoidectomy (right side), tympanostomy, myringotomy and insertion of teflon tubes, stabilization of his scoliosis with insertion of Harrington rods. He was repeatedly evaluated by the pediatric cardiology staff. There were no new murmurs, changes in his electrocardiogram, or progress of the aortic dilatation.

In July 1977, at age 9½ years, the patient helped clean out his parents' garage and then went swimming with his father. While swimming, he developed chest pain and shortness of breath. The pain was described as being mid-sternal in location, crushing in nature, and was also felt over his back. The duration of the initial

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episode was three minutes. The patient experienced several more short periods of pain before being seen in the emergency room at the University of Kansas Medical Center. There was no chest pain during the interview and examination and the vital signs were normal. The electrocardiogram was normal except for a slightly prolonged PR interval. The cause of the chest pain history was unclear, and the patient was discharged from the emergency room with instructions to return if the chest pain recurred.

Chest pain and dyspnea were again experienced upon returning home, whereupon the patient was admitted by ambulance to the KUMC Pediatric Intensive Care Unit. In addition to the previously described chest pain, he also complained intermittently of pain in his head and abdomen. He was anxious, and the dyspnea which had been controlled by oxygen became noticeable again when he was supine. The vital signs were as follows: temperature, 36.7C; pulse, 138 beats/min; blood pressure, 130/70 mmHg; respirations, 48/min. He appeared agitated and in moderate distress. The lungs were clear to auscultation and percussion. Examination of his cardiovascular system showed no cyanosis, normal peripheral pulses, a Grade III/VI systolic murmur, and a Grade II/VI blowing diastolic murmur both heard loudest at the lower left sternal border. Abdominal examination produced no abnormalities; however, he was unable to lie flat for any length of time. His hands had the typical long, thin, lax fingers. Moderate to severe kyphoscoliosis was again noted.

The electrocardiogram was unchanged, but a chest roentgenogram showed moderate cardiomegaly and marked dilatation of the aorta down to the left hemidiaphragm. Six hours after admission, the patient suddenly gasped, became opisthotonic, and had no discernable pulse. Resuscitation efforts were begun promptly but were unsuccessful.

The autopsy revealed an intimal tear at the base of the aortic arch with a huge dissecting aneurysm of the aorta extending down to just above the renal arteries. There was also moderate left ventricular hypertrophy, aortic valvular ring dilatation, and proximal dilatation of the aortic root. The ductus arteriosus was most markedly reduced in size, being only probe patent. A periadventitial hemorrhage of the aorta was present with a hemopericardium of 300 ml, and left hemothorax containing 100 ml of blood respectively.

Discussion

Reported cases of children with Marfan's syndrome and dissecting aneurysms of the aorta are rare. In a review covering reports prior to 1944, only four children under ten years of age had died from aortic dissection.[2] These children were not clearly identified as having Marfan's syndrome. Only one case of a dissecting aortic aneurysm—a 19½-year-old patient—was identified in a series of 36 infants and children with Marfan's syndrome seen between 1945 and 1970 at the Children's Hospital Medical Center, Boston.[3] The youngest child reported with this catastrophe was four years old.[4] A few isolated reports including this complication in a mother and son with Marfan's syndrome have been published.[5,6] Rupture of an aortic aneurysm in a 12-year-old girl following limited exercise testing has also been reported;[7] although the microscopic examination showed extensive degeneration of both elastic and muscular fibers in the media, she had no other findings characteristic of Marfan's syndrome. Other authors have reported similar cases and have suggested this may represent an incomplete or forme fruste type of Marfan's.[8,9]

The first association of aneurysmal dilatation of the aorta and Marfan's syndrome was reported in 1943.[10] Since then the onset of cardiovascular lesions has been demonstrated to occur in infancy.[11–13] In a collection of 11 patients studied by Papaioannou *et al.*, one two-day-old infant had possible dilatation of the aortic root on umbilical venous angiography; the condition was again suspected at one month of age by left atrial injection, and confirmed at four years of age by retrograde angiocardiography.[13] The development of aneurysmal dilatation of the aorta in Marfan's syndrome is believed by some to be an acceleration of normal processes of tissue injury and repair leading to premature weakening of the aortic wall.[14]

Valvular cardiovascular lesions in Marfan's syndrome appear to be more common in children.[3] Of the 36 children studied, 21 had mitral regurgitation, aortic regurgitation or both, while only 10 of 36 had aortic dilatation, including 3 children with combined mitral and aortic regurgitation.[3] In another study, aneurysmal dilatation of the aortic sinuses and mitral regurgitation were coexistent lesions.[13] An autopsy series of 34 patients with Marfan's syndrome found an equal incidence of aortic and valvular lesions, but the number of children included was not specified.[15] The severity of the valvular disease can be quite extreme, as seen in a case previously reported from this institution.[16]

Surgical management of the cardiovascular complication of Marfan's syndrome includes several children who have required either aneurysmorrhaphy[17] or complete replacement of the ascending aorta with a graft.[17–20] In the report by Durnin *et al.*,[21] an eight-year-old girl had a hemopericardium which required

drainage and ultimate replacement of the ascending aorta, although the point of leakage from the aneurysm could not be identified. A six-year-old boy with an acute dissecting aneurysm was managed with surgical repair and survived.[17]

Although there is concern about the underlying aortic tissue in patients with Marfan's syndrome, surgical repair of dissecting aortic aneurysms may be feasible. However, before repair can be undertaken, early recognition of symptoms is critical. Chest pain in adults occurred in 90 per cent of 72 patients reported by DeBakey *et al.*[22] Chest pain as a complaint in children usually does not carry such potentially ominous implications as in adults;[23] however, the primary complaint of our patient was that of chest pain. Therefore, it is imperative that in a child with Marfan's syndrome, dissecting aortic aneurysm must be a diagnostic consideration in spite of its rarity, as noted by the paucity of case reports in the literature.

Summary

Our case of Marfan's syndrome in a 9½-year-old boy demonstrates that a dissecting aortic aneurysm does occur in children. We have briefly reviewed this problem in children, and the onset of cardiovascular involvement of Marfan's syndrome in childhood. The early recognition and prompt surgical management potentially can result in a favorable outcome of this otherwise catastrophic complication of Marfan's syndrome.

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TUESDAY EVENING, MAY 9

PRESIDENT'S BANQUET

Annual Meeting

Kansas Medical Society

May 7-10, 1978

Downtown Ramada Inn Topeka

Burn Update

Management of Scars and Contractures

MANI M. MANI, M.D.; DAVID W. ROBINSON, M.D.;
FRANK W. MASTERS, M.D. *and* LYNN D. KETCHUM, M.D.,
Kansas City, Kansas

SIGNIFICANT changes have been seen in the field of burn medicine the past 25 years. Prehospital care and rescue have been standardized in most parts of the United States. The pathophysiology of burn shock and fluid therapy are better understood now.[1] Topical antimicrobial agents and judicious use of systemic antibiotics have decreased burn wound colonization and death from sepsis. All these factors have reduced the mortality from thermal injuries.

The increase in burn survival rate has not necessarily reduced the late morbidity in the thermally injured victim. Burn wounds heal with scar and the survivors of major burns may have to live the rest of their lives with hypertrophic scars and disabling contractures *(Figure 1)*.

Partial thickness injury to the skin should heal by epithelialization from the epithelial cells retained in the deeper parts of the dermis. This type of healing should be expected within two or three weeks after the injury *(Figure 2)*. In full thickness injury, healing can occur only by creeping epithelialization from the edges of the wound and by scar contracture. In instances where full thickness damage is extensive, the individual may not survive long enough to achieve this form of healing, and skin-grafting will have to be done to obtain epithelial coverage.

In the healing process of both partial thickness and full thickness injuries, collagen is laid down under the epithelium. The amount of collagen formed in the scar varies with the individual and is affected by age, race, time required for epithelialization, and the site of the injury. The scar is very vascular and individual strands of fibrous tissue appear to be laid on without orientation. Linares[2] has shown that the fibrous tissue in the early scar appears to be in whorls, and that this pattern changes with maturation, immobilization, and elastic compression *(Figures 3 and 4)*.

It has long been known that splinting will reduce the activity and hypertrophy of the burn scar. This has the disadvantages of joint stiffness, disuse atrophy of the muscles, and demineralization of the bone. Elastic

Methods of treatment for minimizing scars and contractures in the thermally injured are discussed and illustrated.



Figure 1. Severe hypertrophy and contractures of arm following full thickness burn.

Address reprint requests to Dr. Mani, KUMC, 39th & Rainbow Blvd., Kansas City, KS 66103.



Figure 2. Healing of partial thickness burns from the remnants of epithelium in hair follicles.



Figure 3. Burn scar hypertrophy treated successfully with pressure gradient therapy.



Figure 4. Same as Figure 3.

compression with the use of elastic bandages is effective, but it is difficult to maintain sustained and adequate compression for prolonged periods of time. It is realized now that compression needs to be maintained for almost 24 hours each day, and that the pressure should be around 25-35 mm mercury. In the past few years, custom fitted, pressure graded garments have been made available. The use of these garments has helped to overcome some of the problems of compression therapy for burn scars. Each patient must be measured and custom-fitted as soon as the burn wounds have healed and the patient is able to tolerate the pressure over the recently healed areas. The pressure graded therapy must be maintained for at least 9-12 months, or as long as the scar shows evidence of activity or hyperemia. Patients are rehabilitated to go back into their pre-burn activity wearing these garments. Gloves, sleeves for the arms, vests for the trunk, face masks for the scalp *(Figure 5)* or face, and support hose for the lower extremity and lower trunk are available. The first set of garments available to patients come with zippers. This avoids the shearing strain on the recently healed epithelium. Once the patient has been wearing these garments for three to four weeks,

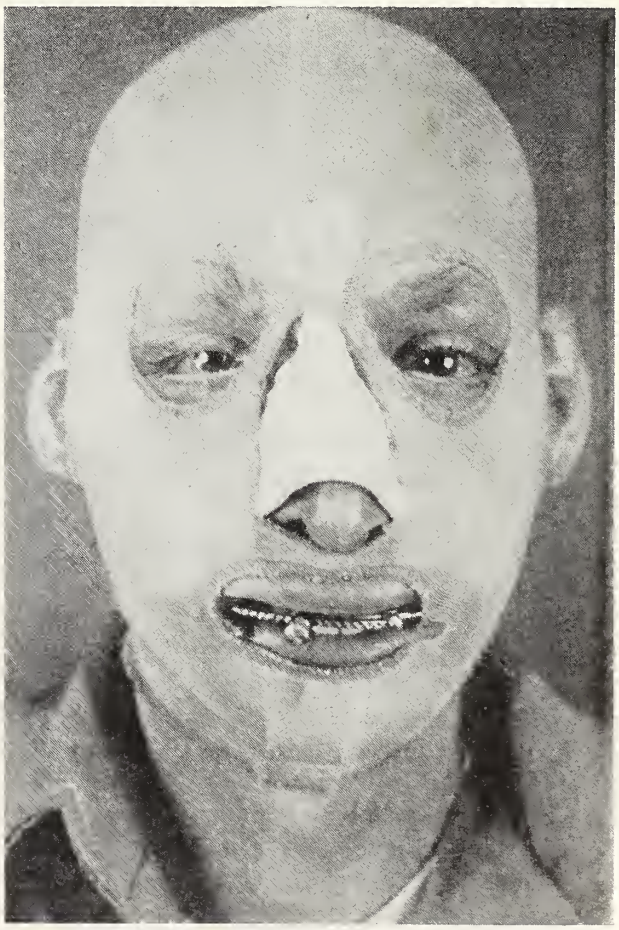


Figure 5. Face mask, nasal splint, and appliance to prevent microstomia in a patient with severe facial burns.

the second set of garments is ordered (this without the zipper). Patient acceptance of such garments may be a problem initially, but once the patient realizes that the garments give him mobility and reduce the amount of discomfort, there is very little difficulty in persuading the patient to wear this for nearly 24 hours/day.

Static and dynamic splinting is another method of reducing and preventing burn scar contractures. Static splints also have disadvantages, such as joint stiffness, and patients are instructed to remove the splints and go through the full range of motion at least three times/day. Dynamic splinting using elastic traction also has some of the disadvantages and will have to be removed and reapplied periodically. Neck splints are extremely beneficial in treating and preventing neck scar contractures. Web spacers for thumb and fingers must be worn outside the elastic compression garment. The use of splints in active patients is usually restricted to night-time, when the patients are resting or sleeping.

Summary

Many more patients survive major thermal injury now than in the past. This has increased the morbidity in the form of late burn scar hypertrophy and contractures. Pressure graded therapy has definitely reduced the late morbidity of these injuries. The need for late reconstructive surgery can be reduced by the early and judicious use of elastic compression. Patients can be made functional much earlier and this in turn reduces the cost of repeated hospitalization and loss of work days.

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Month in Washington

(Continued from page 84)

reported to be three to ten times higher with blood from paid donors versus blood from volunteers.

Dr. Kennedy said that 10,000 to 30,000 cases of post-transfusion hepatitis occur each year in the United States with at least 400 deaths resulting.

Medical Deductions

President Carter has proposed in his tax message to the Congress changing medical deductions on personal income tax by combining the separate deductions for medical expenses and uninsured casualty losses into a new "extraordinary expense" deduction. The new deduction would be available only to the extent that these items together exceeded 10 per cent of the adjusted gross income.

The American Medical Association has before the Congress a proposal (H.R. 5188) that would permit a taxpayer to deduct the full amount of medical and drug expenses paid for the medical care of himself, his spouse, and dependents.

Project Integrity

More than 2,400 doctors and druggists providing subsidized health services to needy persons have been identified as having "patterns of practice indicating a likelihood of fraud and abuse," HEW Secretary Califano has said. He announced new details of Project Integrity, a program of HEW searching for corruption in subsidized medical care.

HEW has issued regulations, required under 1977 anti-fraud legislation, that set requirements for states creating fraud and abuse control units to monitor the federal-state Medicaid program. The units should operate separately from the agency administering a state Medicaid plan, have the capacity to prosecute fraud or refer allegations of fraud to prosecutors, and investigate complaints from patients in nursing homes and mental institutions.

If states create such units, HEW will reimburse them for 90 per cent of their operational costs, a government spokesman said.

Califano said Project Integrity has screened the billing claims of all 275,000 Medicaid physicians and pharmacists "and identified over 2,400 with patterns of practice indicating a likelihood of fraud and abuse." More than 450 of the 2,400 doctors and druggists are being investigated for potential Medicaid abuses. Another 400 are undergoing "detailed field checks for potential criminal fraud," Califano said. Cases involving about 200 have been closed as not warranting further investigation. The other cases are still in the investigation pipeline, Califano said.

HEW also plans to review another 44,000 cases where preliminary information has indicated the possibility of fraud and abuse.

Buy U.S. Savings Bonds

Tenets of Treatment

Critical Care of the Pediatric Patient

DAVID R. STEWART, M.D.,* *and* BETSY MALLEY, R.N.,†
Kansas City, Kansas

"CHILDREN are not just little adults" is a phrase which clearly applies to the critical care of children. The nuances of fluid and electrolyte balance are nowhere more critical than in the care of the seriously ill infant, and the margin of error in these patients is much narrower than in the adult. On the other hand, children are often more resilient than adults because they do not have generalized diseases such as arthrosclerosis and, unless directly affected by an acute process, pediatric hearts, lungs, kidneys, and brains are healthy and strong.

The Intensive Care Unit

The optimum care of the child is accomplished in an area specifically designed as pediatric, and by personnel trained in the care of children. Sick children and sick adults do not mix well, and the Pediatric Intensive Care Unit (PICU) is the best place for a critically ill child. Such a unit should be staffed by a minimum of one nurse for every two patients and, in the case of the very ill child who requires constant attention, one nurse per patient. At all times there should be at least one nurse in the unit who has had additional training in the critical care and resuscitative skills. Such experience can be gained through specific courses such as the one taught by the School of Nursing at Kansas University Medical Center.

The PICU should be designed to have at least 120 sq ft allotted for each bed space. This allows room for extensive monitoring equipment, portable x-ray machines, and working space for special diagnostic or resuscitative procedures. There must be at least six electrical outlets per bed space, and ceiling or wall suction as well as oxygen lines must be provided.

Monitoring

Currently available equipment allows even the smallest of neonates to be carefully monitored, but at no time should the machine be allowed to replace the expertise of the medical staff. Even the most sophisticated of monitors can serve only as adjuncts to good medical care. The two parameters most commonly recorded are the ECG and the arterial blood pressure. Unfortunately, the latter measurement involves invasive techniques, usually percutaneous catheterization of the radial artery, and presents some hazards. Prior to using the radial artery of a child, which may be no larger than the diameter of the catheter, an Allen's test should be performed. If appropriate collateral circulation is not demonstrated, the artery should not be used. With the establishment of a radial (umbilical) artery line, a source of arterial blood gases is available. However, when dealing with infants and children, one must remember to be frugal with the amount of blood withdrawn for laboratory work. Many a child has experienced a hematocrit drop of 4-6 points from repeated laboratory sampling. Do not be tempted to use the often large femoral vessels as sources of blood for laboratory samples or as catheter routes unless it is absolutely essential for survival. The risks of aseptic necrosis of the femoral head, traumatic AV fistula, false aneurysm formation, and sepsis militate against the use of these vessels for any purpose in a child. Except in the severely injured patient or in one who is actively bleeding, we have not found it essential to use CVP lines.

Arterial blood pressure and urine output combine to give a very good estimate of circulating blood volume. If a CVP catheter is needed, it is best placed through the brachial or external jugular vein. Subclavian vein catheterizations are associated with a higher risk of venous damage, and also with a significant incidence

Basic procedures for the care of critically ill children are discussed and some guidelines indicated for certain types of treatment.

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of pneumothorax due to the higher pleural apex in children. There has been an increase in the use of the Swan-Ganz catheter in our unit, generally in patients with pulmonary trauma or "shock lung." The smallest of these catheters is a size 5 French which can be used in the average two- or three-year-old child. Use of Foley catheters should be avoided. Most children have no impediment to urinary flow and will void frequently if well hydrated. Indwelling catheters are an invariable source of urinary tract infections if left in place longer than 72 hours and often cause meatal stenosis in boys as a result of local irritation. One new type of monitoring is that of intracranial pressure by means of a trephine-type of screw. We have used this information when treating children with Reye's syndrome, and others have used it during the recovery phase of a severe head injury.

Resuscitation, Fluid, and Electrolyte Management

Ringer's lactate can be utilized to resuscitate children after trauma or other situations resulting in hypovolemia. As much as 20 cc/kg can be given rapidly and safely, but care must be exercised to prevent development of a hyperosmolar state in neonates and infants whose kidneys have a diminished concentrating capacity and do not excrete sodium as well as in older children. As a general rule, $D_5\frac{1}{4}NS$ is a satisfactory maintenance intravenous fluid for children. Colloid replacement with albumin or plasma can be safely given at a rate of 10cc/kg; this can be increased to as much as 15cc/kg if the patient is severely protein depleted. Packed red blood cells can likewise be given at a rate of 10-20cc/kg depending upon the hematocrit of the patient. If the patient has a nasogastric tube in place, the drainage should be replaced using one-half normal saline containing 20 milliequivalents KCl/liter. All critically ill infants and children should have daily measurements of electrolytes and BUN; if they are receiving nephrotoxic drugs such as Gentamycin, a serum creatinine test should be done at least twice a week. A urine output of 15-20cc/kg/day is the lowest acceptable amount in a child.

Careful attention to detail when treating the critically ill child will be rewarded by recovery of the patient. To paraphrase Dr. Willis J. Potts: The infant "with no language but a cry," and the child with no words to express the desire to be well and normal, ask that we make available to them the benefits of increased knowledge of the essentials of critical care.

Telling It Like It Is

Socialized Medicine at the Grass Roots

Dr. Cree, Dr. Nestor, Lt. Spears, Grace; Randegar, Notedge, Thaylor—people you've never met, places you've never been? Don't be too sure. These are the fictional names and places utilized by A. S. Reece, M.D., "to protect the unfortunate and also the guilty" in his recently published book, "Socialized Medicine at the Grass Roots." The story, says Dr. Reece, is all true. In essence, it relates the life experience of Sam Cree, M.D., picking up on his life as he establishes his practice simultaneously with the stock market crash of 1929, and following the details of his everyday world into the present with its Medicare and Medicaid involvements.

Dr. Reece has printed 500 copies of his medical saga, and has advised that they are available without charge to readers of the *Journal*. Requests should be addressed to:

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Gardner, KS 66030

Circulatory/Respiratory Dynamics

Continuous vs. Intermittent Ventilation

HUGH S. MATHEWSON, M.D.,* *Kansas City, Kansas*

MECHANICAL VENTILATORY SUPPORT involves the adjustment of pressure, volume, and time parameters of external respiration, and the administration of supplemental oxygen. This is required under the following circumstances:

1. When failure of central respiratory cycling has occurred. The problem may be slowing of rate, as seen in narcotic overdose and some cases of increased intracranial pressure; or it may be a reduction in tidal volume, as occurs in barbiturate poisoning. Either causes a critical reduction in alveolar gas flow.

2. When neuromuscular failure renders the patient too weak to breathe. The performance of ventilatory work is curtailed, and positive pressure is required to supplement tidal exchange.

3. When compliance loss raises the pressure required to deliver an adequate tidal volume, and the ventilatory work load is increased.

4. When a rise in airway resistance necessitates increased pressure to maintain alveolar gas flow, thus augmenting ventilatory work.

Continuous Ventilation

Treatment of respiratory failure involves the application of a device which delivers positive pressure in the inspiratory phase, then opens to the atmosphere, allowing exhalation to occur. In some instances it may be advisable to maintain positive end expiratory pressure (PEEP) throughout the respiratory cycle to increase the functional residual capacity and to prevent premature airway closure. In all cases, however, the entire respiratory minute volume is provided by the machine.[1]

The mechanical ventilator usually employed for continuous support is a flow generator which delivers a preset tidal volume at whatever pressure is required up to an adjustable limit. If inspiration is triggered by a timing device on the machine, ventilation is said to be *controlled,* that is, both volume and rate are fixed at predetermined values. If inspiration is triggered by the negative inspiratory pressure exerted by the patient, ventilation is said to be *assisted;* volume is predetermined, but not rate.

* From the Department of Respiratory Therapy, KUMC.

Controlled ventilation is satisfactory only if the patient yields readily to the imposed pattern. Often there is considerable asynchrony ("fighting the ventilator") when the machine is first connected to the patient. Sedation with diazepam (incremental doses of 5-10 mg) or suppression of respiratory drive with morphine (doses of 5-15 mg) may be effective, but some cases require the additional use of a muscle relaxant. It can often be questioned whether the pharmacologic depression necessary to achieve ventilatory control yields benefits which outweigh the risks. Sufficient stability of respiratory parameters may be possible if the patient is allowed to trigger inspiration. Control of rate is then lost, and continued administration of high tidal volumes is likely to cause overventilation. Sudden shifts of $PaCO_2$ to levels below 30 mmHg may produce alkalemic neuromuscular disturbances, hypotension, and cardiac arrhythmias. However, with gradual changes, there is little or no interference with vital functions, and patients generally tolerate mild states of alkalosis without adverse effects.

Intermittent ventilation is explored as an option for patients with some capacity for spontaneous breathing.

Most awake patients accommodate to assisted ventilation better than to complete control. Circulatory reflexes are better maintained with the patient free of the depressant effects of drugs. If spirometric values are reasonable and the pH is stable at 7.35-7.55, assisted ventilation is usually the mode of choice. Communication with the patient is facilitated, and sensorial evaluation is more accurate if sedation can be held to a minimum. However, PEEP is less effective, and uneven ventilation may occur if tachypnea develops. It is customary to employ the volume ventilator as a guarantor; that is, to allow patient triggering, but to preset a controlled rate somewhat less than the spontaneous rate. Then, if the patient fails to breathe, the machine will cycle on at time intervals adequate to maintain ventilation.

Intermittent Ventilation

It may be unnecessary to assist or control ventilation continuously. Spontaneous breathing may be sufficient if occasionally supplemented by a machine breath. Fighting the ventilator can largely be eliminated, and the patient's own ventilatory capability can be continuously evaluated. The intermittent breaths may be controlled, that is, delivered at specified time intervals (intermittent mandatory ventilation), or they may be augmented assisted breaths (intermittent demand ventilation). Present technology has provided a mechanical continuum between controlled and spontaneous ventilation which allows a complete spectrum of ventilatory patterns from which to choose the one most appropriate to the needs and capabilities of the patient.

Intermittent mandatory ventilation (IMV).[2, 3] A volume-cycled ventilator can be fitted with an attachment incorporating a one-way valve which directs gas flow either from the machine or from a separate source of metered air, providing an oxygen supply that can be drawn upon for spontaneous breathing. The latter delivers a relatively high flow, and the patient ventilates spontaneously from essentially a semi-closed nonrebreathing system. At selected intervals, a preset volume of gas is delivered from the machine, closing the one-way valve and inflating the patient's lungs, guaranteeing a minimum respiratory volume per minute.

The machine cycle intrudes to some degree on the patient's spontaneous breathing; some individuals find this annoying and uncomfortable and may fight the machine. Also, the preset volume is usually large; if superimposed at the height of spontaneous inspiration, excessive pressures may be generated (but this can be controlled by the pressure limiting device).

A recent improvement in ventilator design is termed Synchronized IMV (SIMV). The preset volume from the machine is delivered on initiation of inspiratory effort by the patient, this being monitored by a pressure sensor or rate counter. If no inspiratory effort occurs, the volume is still administered on signal from a timer.

Intermittent demand ventilation (IDV).[4] This simulates a maneuver familiar to anesthesiologists, providing a bag squeeze with every third or fourth spontaneous breath. The machine is set to operate in the assist mode, with only selected assisted breaths delivered to the patient. The other cycles are exhausted into the room, and the patient breathes only his natural tidal volume from a gas reservoir. IDV can resolve some problems of patient-ventilator asynchrony, and is less obtrusive than IMV. An alarm system is necessary to provide a minimum respiratory rate should the patient become apneic.

Uses of Intermittent Systems

To qualify for intermittent ventilation a patient must be able to support gas exchange by spontaneous effort, at least for brief periods. The modality is primarily intended to prevent progressive exhaustion, due either to increased work demand or incoordination of the respiratory musculature. It is not intended to "catch up" on ventilatory deficits incurred during spontaneous breathing intervals. Some guidelines are suggested in dealing with each of the four categories of respiratory failure:

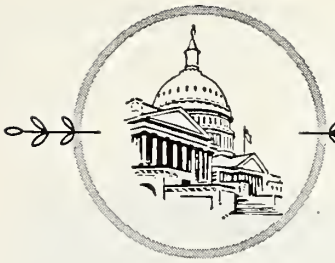
Central respiratory failure. The patient should be capable of moving tidal volumes of 7 ml/kg at rates of 10-30 breaths/minute. Ataxic or periodic breathing is a relative contraindication. Should an unexpected apneic interval occur, controlled or guaranteed ventilation must be reinstituted without delay.

Neuromuscular failure. Intermittent ventilation can be of great value during the recovery phases of polyneuritis, myasthenia gravis, or cholinergic crisis. However, the minimum criteria for weaning should be met. The patient should be capable of demonstrating a vital capacity of 10 ml/kg and a negative inspiratory pressure of 20 mmHg. Blood gas values must be stable throughout the period where gradual reduction of the mandatory rate is carried out. Sufficient support to prevent episodes of hypoventilation is necessary, and withdrawal can require several days.

Compliance loss. An essential criterion is demonstrable decrease of the work of breathing. Resolution of stiff lungs, relief of lung compression, or restoration of thoracic integrity following chest wall injury may be supported by gradually decreasing intermittent ventilation. Splinting due to pain usually requires analgesia sufficient to permit continuous ventilation before an intermittent system can be tolerated. Patients with morbid obesity undergoing shunting operations may need extended ventilatory assistance in the postoperative period; an intermittent system often provides a satisfactory solution. Again, minimum criteria for weaning should be satisfied, and stability of respiratory exchange verified by serial blood gas determinations.

Increased airway resistance. Fewer suitable cases can be drawn from this group. Severe asthma patients need continuous support during the acute episode.

(Continued on page 139)



Socio- ECONOMICS

Preparing a Patient Information Booklet

Ed Note: This is the fifth in the series of articles prepared by the AMA Department of Practice Management, Division of Medical Practice. For other articles on this topic, see the following issues: November, 1977, December, 1977, and January, 1978.

The subject of practice management has become more critical in recent years. To assist the physician in a smoother operation of his office, the Department has produced workshops for medical assistants dealing with such aspects as collections, public relations-telephone techniques and office management, as well as practice management workshops for the new physicians. The Kansas Medical Society Executive Office will present such workshops upon demand by members.

RIGHT NOW YOU probably have a patient who is wondering:

why your medical assistant asked for his symptoms on the phone when all he wanted was a simple appointment;

exactly what a physician in your specialty does;

why her "other doctor" doesn't schedule appointments six months in advance;

if your assistant is going to ask him to pay now for today's visit;

if she should call you at home if the baby is sick at 8:00 PM.

Whether you are a solo family practitioner or a specialist in a group practice, you will find that a patient information booklet is a good way to answer these and other questions your patients have. It is an effective way to communicate. Consider the benefits:

1. You can let patients know what to expect from you and your office staff *before* questions or problems arise. This can only improve patient relations.

2. Patient information booklets have been shown to be effective in reducing unnecessary telephone calls. In some practices booklets have cut incoming phone calls by as much as 20 per cent. Think of the saved staff time!

Where to Begin

First decide what areas your booklet will cover. Topics most often discussed are: specialty and philosophy of care; what to expect on a first visit if you are a specialist; appointments; returning telephone calls; billing; insurance; hospital privileges; reception room; staff services. Some physicians also include a section on general clinical information and recommended first aid procedures. Others have added a map showing the office location. What you decide to include depends on you, your specialty, and your style of practice.

Your medical assistant and office staff may be asked questions repeatedly that you never hear, so they will be able to supply helpful ideas. Also keep in mind that the most effective patient information booklets are written using "I" or "We" and are conversational in tone. As for style, it is always a good idea to show the patient how following a policy or office routine benefits him or her. For example, it is better to say, "we ask that you call and cancel an appointment as far in advance as possible so we can give this time to another patient," than "please call and cancel an appointment if you can't make it so the doctor's valuable time isn't wasted." Booklets taking a "Thou shalt not . . ." approach are rarely effective and are most often offensive to patient-readers.

What Will It Cost

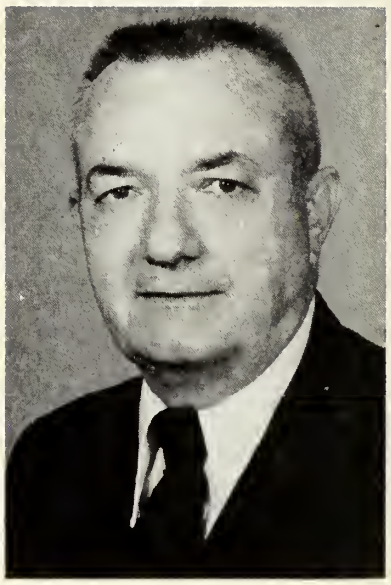
The cost of developing a patient information booklet is small. Your "first edition" can simply be typed on a standard 8½ x 11 in. sheet of paper, which is folded in half, booklet style. You can take it to a duplicating service who can mimeo the sheets for a low cost on colored paper. Later you may decide to have the booklet set in type at a local printer. Checking with several print shops for cost estimates and choosing a type face that elderly patients can easily read is advisable.

Booklet Distribution

This is the last step in implementing a patient information booklet, but it may be the most important. It is best if you or a member of your staff gives the booklet out and says: "We're sure as a new patient you have some questions about our practice that you may not have asked today. We may have answered your questions in this booklet, please *read it, keep it, and refer to it.*"

(Continued on page 131)

The President's Message



This is the last issue of *The Journal* this year in which my thoughts, exhortations, and reflections will appear. The past year has gone by suprisingly quickly, and it hardly seems possible that the annual meeting, this year in Topeka, is just weeks away.

This year as President of the medical society has given me an opportunity to fully see the dedication and hard work of your officers, committee chairmen and members, office staff, and all the others who have made involvement in Kansas Medical Society activities one of their priority items. It is the ongoing efforts of you and your colleagues that keep the medical society functioning from year to year, and the comments, criticisms, and input from you during this past year were truly appreciated.

The Legislature is still ruminating over several measures of interest to us and, as usual, the final word on most of these issues will not be in until final adjournment in mid-April.

A significant Medical Society effort to identify and rehabilitate physicians disabled through drug or alcohol abuse was developed and implemented this year, and we are optimistic of its potential impact in the future. This newly created Physician Performance Committee will commence full operation after House of Delegates' final approval of the plan of action.

Another important program recently implemented is the joint "voluntary effort" of the Kansas Medical Society and Kansas Hospital Association to curb the rapidly increasing rate of hospital and other health care costs. We have already had several meetings with the Hospital Association, and a broad-based and diversified steering committee is being assembled to help guide this voluntary effort as it attempts to deal with the important task before it.

One of the most enjoyable privileges of being President is the opportunity to visit all of the Council districts in the state and discuss current issues with the members. We will always remember the hospitality, courtesy, and kindness you showed us on our travels over the state this past year.

This year as President of your medical society has afforded me the opportunity to recognize one crystal-clear fact which stands out above all others. I cannot emphasize too strongly the importance of you taking an active interest in the events which are shaping the future of our profession. There is no end to the number of planners, agencies, and bureaucrats standing in the wings waiting to shape and mold the practice of medicine and delivery of health care as they see fit. The continuing trend of both federal and state government intervention in the practice of medicine is growing at an alarming rate, and the credibility of the profession is constantly assailed and challenged. It matters not that we find the ever-increasing intervention into the practice of medicine repugnant. The current climate necessitates our individual involvement whether it be political action, serving on an HSA board, working with PSRO, or by actively supporting your medical society. The single biggest threat to medicine today is the inability or unwillingness of individual physicians to take an active part in the events which are shaping the future of the profession.

Serving you this year has been a gratifying and rewarding experience, and I am thankful to have had this opportunity. Thank you!

Fraternally yours,

John D. Huff M.D.

President



Editorial COMMENT

Metamorphosis

This is probably an exercise in temerity, since most of those to whom these presents shall come are in a better position to state the case—and moreover would consider themselves so. However, with typical journalistic fatuity, we intend to record one of the more interesting medical phenomena of the times, the rise and reign of the family practitioner. And we hasten to say, before we lose that audience, it's kindly meant.

The generic term is "general practitioner" but, since the art is long, even that is of fairly recent coinage. It was a product of the Age of Specialization, since prior to that there was no need for the designation. There were numerous distinctions—the herbalists, the homeopaths, the eclectics, and so on—but to identify the physician as providing the general service had no meaning since it was assumed for all.

The emergence of the surgeon as a primary medical mutation established the non-surgical colleague as a supplier, necessary but inherently subordinate. Thus, the term general practitioner was devised to indicate the total general medical function—but not, of course, including surgery. A few of the malcontents chose to resist and, relying on an expanding repertoire of laboratory esoterica and carrying their stethoscopes at the ready, they called themselves "internists" (leaving the outside for the dermatologists, apparently). Others followed the trend. It was easy, for example, for the obstetricians to establish their territory, since this practice appealed primarily to certain oddballs who didn't care what hours they kept so long as the patient did the work. Then the pediatricians took over the babies (vitamins and immunizations having been invented), keeping them until they were having their own. Increasingly, the general practitioner became defined by the things he was not supposed to do, and, ironically, the more his role was defined the less he had to be general about. Inevitably, he felt he was left with the undesirable elements of practice —less remunerative too, unless he pedalled a lot faster. (There was a time—long since gone, of course—when a cozy exchange of monies between surgeon and general practitioner helped resolve the economic inequities, but someone decided this was equivalent to Mom slipping ersatz apples into the pie, and blew the whistle.)

Lest the gp forget his place in the medical pecking order, his medical education consisted of instruction in a variety of specialties by specialists. In his rotating internship (presumed to provide general experience), he was handed over to other specialists, none of whom acknowledged that any of the others had any claim on the intern's mind or body. This was the end of his formal education, permitting the specialist's attitude that he was either too poor or too dumb to go further. Once in practice, he found his postgraduate courses provided by (and largely for) specialists selected on the basis of their distance from general practice. Occasionally, one would assure him with studied indulgence that this or that procedure could be done "even by a gp," but somehow when he got home none of the specialists had heard about that part—unless it was at night or a welfare patient.

Patients were divided. Some enjoyed the self-bestowed prestige of being patients of a specialist, but others claimed undying fidelity to their general practitioners. But overall, the change in medical care delivery with a disproportion of specialists meant that the first line of medical care was left increasingly understaffed. The patient became less concerned with whether the general practitioner was doing things the specialist objected to or the specialist was giving general care outside his formal limits. He was concerned about getting *some* attention.

The general practitioner's heroic effort, which finally began to break the pattern, was made up of one part boot-strap tugging, one part public relations, and one part education. He came to the conclusion, first, that he was in danger of extinction and, second, that no one

else was going to do anything about it so he had better get to work and change the situation. His first effort, understandably, was an attempt to improve his public image and he made a try at being known as a "generalist"—which fortunately died aborning. Then a couple of changelings were spawned along the way. One was designated the primary care physician. This didn't really solve anything though since, even if it suggested to the patient the idea of immediate care, it still implied he would have to go on to someone else after the initial attention. (And the obstetricians were quick to call themselves primary care physicians for the female population, thereby cornering 50 per cent of the market right off.) About this time, the emergency physician emerged. Since the hospital administration had despaired of getting the medical staff to devise effective coverage of the emergency room (the specialists again), it began to hire moonlighting physicians (usually from some nearby government installation) to do the job. Out of this evolved the emergency room physician even as the emergency room became an outpatient clinic (a source of revenue the hospital was not about to stop).

The turning point really came with the amalgamation of this formless mass of general practitioners into an academic entity, organizational first, then educational. With the establishment of postgraduate training requirements and a testing procedure leading to certification of academic qualifications, a degree of parity with his specialist colleagues was achieved and the image struggle was largely over. The attendant publicity and change of name to "family practitioner" assured his identification as being primarily concerned with all members of the family—and as patients first rather than examples of specific disease. The student arriving at medical school with the announced intention of going into family practice was not shunted to the side door but greeted with professorial smiles and sought by lay committees seeking to sign him to a contract for future service. The formalization of postgraduate training into a family practice residency and the well-nurtured implication of dedication to humanistic principles even led to Sunday supplement feature stories and TV talk show appearances. (Of course, the process was aided by certain economic facts of life. House staffs were now commanding sustaining stipends, the lack of which had played no small part in diverting many new graduates of earlier days immediately into the world of general practice.)

So it is that this tribute to the family practitioner (ne general practitioner) finds its way into the issue of this periodical which is devoted to products of that temple to specialization, the Medical Center (Kansas City version, that is). It celebrates not only the reissue of this most basic of physicians in a rehabilitated form but, more significantly, the incorporation of the process into the function and curriculum of the teaching institution, assuring at once the equality of the practitioner products and the viability of the concept.

As is usually the case, the success of such an effort, for all its timeliness, can be attributed largely to the efforts of certain individuals—in this case, chiefly Jack Walker and George Burket (and more recently Larry Miller in Wichita), who have catalyzed the academic and practical elements of the effort, assuring the professional educational continuity even as the family practitioners assure its professional service effectiveness. Dr. Walker and Dr. Burket have been involved in this phoenician enterprise since before it was popular to be so. They have applied their energies and talents not only to upgrading the family practitioner's status, but to the associated efforts of increasing physician supply (both by import and retention) and improving distribution.

This relationship between the family practitioner phenomenon and the increasing attention to the problems of physician supply and distribution deserves emphasis particularly in a state such as Kansas, which has a significant problem in providing medical care to areas of relatively thin population—and particularly because the integration of these elements has always been one of the essential efforts of Kansas medicine.

There is more than a little similarity between the role of the family practitioner who brings together the essentials of medical service and makes them available to a broad clientele, and the organizational effort to combine these health care delivery elements and make them available to the citizens of Kansas. The laity has gained a new appreciation for the former. The latter must be clarified to the same laity which is ill-advised to attempt to resolve by legislative pressure those problems (others as well as medical) of service which cannot be met except with the concerned and willing efforts of those who will be doing the serving.—*D.E.G.*

CME Relicensure in Kansas

Reporting Date Is Near—July 1, 1978

From and after July 1, 1978, every licensee rendering health care service in the state is required to submit with his or her request for license renewal evidence of satisfactory completion of a program of continuing education.

The Board of Healing Arts will accept completion of the AMA Physicians Recognition Award or the CME certification programs of those specialty societies recognized as equivalent to the Physicians Recognition Award.

Upon completion of the CME requirement, the AMA or the specialty society will notify the Kansas Medical Society office. A certificate will be mailed to you prior to your licensure renewal date. Please retain one copy of the certificate for your files and forward one copy to the Board of Healing Arts with your license renewal form.

Any physician licensed after July 1, 1975, will have three years to complete 150 hours of continuing medical education for licensure renewal.

If an applicant for renewal will not render health care service in Kansas, the applicant shall submit with his or her request for license renewal an affidavit stating that he or she will not render such health care service during the term of the license.

The Board of Healing Arts may grant a waiver for a specified period not to exceed eight (8) months to a physician who has suffered an illness or accident making it impossible or extremely difficult to comply with the CME requirement.

The Dean's Letter

(Continued from page 88)

practicing physician; (2) continue the development of our research programs, many of which are nationally recognized; and (3) improve patient care.

I want to emphasize our continuing commitment to excellence in patient care. Everybody expects this from an institution and I will spare no effort to make our patient care programs exemplary. I will be calling upon the 7,000 plus people who work or go to school at the University of Kansas Medical Center to attend to even the smallest details to enhance patient care. To help accomplish this goal an institution-wide "I Care" campaign has been instigated at the University of Kansas Medical Center.

I realize my new job represents an enormous challenge, but I accept it. I will count on all of you to let me know how we are doing through the reports you receive from patients you refer to the Medical Center.

Again, I welcome your suggestions.

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PHYSICIANS IN HISTORY

The Declaration of Independence contains the signature of four physicians. Courts-martial for malfeasance in the operation of a military hospital during the American Revolution added infamy to the names of two others. Chance called still others into the service of their fellow man and their country where history has not accorded all participants equal prominence.

James Hutchinson belongs to the inconspicuous group. He left Philadelphia in 1774 at the age of 24 to study medicine under John Fothergill in London. In 1777, Benjamin Franklin, then serving as American Minister at the French court, directed him to return home immediately by way of France. There, Franklin gave Hutchinson sensitive information he had gathered for Congress. As Hutchinson's ship sailed into sight of the American coast, a British cruiser gave chase and attacked. Under open fire he gathered together his secret dispatches and fled to shore in an open boat where he witnessed the capture of his ship and the loss of an invaluable library of medical books collected in France and England.

James Hutchinson continued unobtrusively and loyally to serve his country, his profession, and his fellow persons until he died of yellow fever in 1793. Where many fled from epidemics, Hutchinson remained, treating everyone with equal concern. History says he contracted the disease while attending an impoverished elderly woman in her neglected home.

To some, James Hutchinson died of yellow fever; to others he died of overzealous treatment—purging. John Shippen attended him at the time of his death and later saw fit to publicly defend his skill.

Early American physicians served in a climate harboring many adverse opinions about the doctor's ability, motives and practice. Even Franklin liked to tell the story about an epidemic in Barbados, where only one person survived while medical supplies lasted, but only one died after use exhausted them.

A clergyman of keen observation and vast exposure to the sick and dying said patients seemed to fare better in the hands of the unorthodox than the orthodox physicians.

Does this carry a familiar ring? To those following the ebb and flow of contemporary legislative thought and action, it should. From ancient to modern times, the body of medical knowledge has multiplied, and the practice of medicine has reached a phenomenal degree of precision; yet there are those who would lay the profession aside for a lesser service. Laetrile stands as a prime example.

Physicians must remain politically active, not for themselves alone, but for the sake of the people in the community they serve. You might say, this is their professional responsibility.

Become active, join and support KaMPAC today!

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American Academy of Family Physicians: 6.3 hours of Prescribed Credit.
Registration Fee: None

For program announcement and information, write: DIVISION OF CONTINUING EDUCATION, The University of Kansas College of Health Sciences and Hospital, Kansas City, Kansas 66103.

Preparing a Patient Information Book

(Continued from page 125)

This is much more effective than simply having copies available in the reception room. Some patients may decide *National Geographic* or *McCall's* looks more interesting! Some specialists mail their booklets in advance of a first appointment, which is an especially good idea if the patient has a one-time problem. And, it is advisable to distribute the booklet to established patients when they come in for an office visit. To sum up, your patients will think the booklet is important *only* if *you* do.

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Percutaneous Nephrostomy

(Continued from page 106)

liative urinary diversion for the terminal cancer patient. The two cases presented are representative of our experience with this technique. We feel percutaneous nephrostomy is the procedure of choice for temporary or long-term palliative urinary diversion, if a ureteral stint cannot be passed from below and if definitive surgical correction of obstruction is not feasible. A major operation solely for nephrostomy can usually be avoided.

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Learning Disabilities. April 13-14, 1978. A multi-disciplinary faculty will address the national goal of comprehensive services for all handicapped children by 1980. Lawrence Bradford, Ph.D., will be the workshop leader. Ernest L. Boyer, Ph.D., U.S. Commissioner of Education, will be the keynote speaker.

Physician's and their Families: An Experience in Communication. June 18-23, 1978 and August 13-18, 1978. Workshop will be held at the YMCA of the Rockies, Estes Park, Colorado. This workshop will explore the developmental phases of family life, provide understanding of family communications and reevaluate the implications that medical practice has on family life.

The Three C's of Psychiatric Nursing: Compassion, Conceptualization, Competency. April 28-29, 1978. The role of the nurse in the psychiatric setting will be explored. Norine Kerr, R.N. will be the workshop director.

CME credit available.
All workshops, unless noted, will be in Topeka, Kansas.

For further information contact: June Housholder, Division of Continuing Education, The Menninger Foundation, P.O. Box 829, Topeka, Kansas 66601. 913/234-9566, ext. 3685.

Obituaries

JUSTIN A. BLOUNT, M.D.

Dr. Justin A. Blount, 83, died January 22, 1978, in Larned. He was born November 6, 1894, in Bronson.

Dr. Blount was graduated from the University of Kansas School of Medicine in 1921. He practiced for ten years in the Burdett area prior to moving to Larned in 1931, where he practiced until his retirement in 1972.

Survivors include his wife and two children. Memorial funds have been established with the Leukemia Foundation and with the AMA Scholarship Fund.

EDWARD L. FITZGERALD, M.D.

Dr. Edward Lawrence Fitzgerald, 70, died January 13, 1978, in Morgantown, West Virginia. He was born January 15, 1907, at Jamestown, Kansas.

Dr. Fitzgerald received his medical education at Marquette University and practiced in Hutchinson from 1929-1971.

Survivors include a daughter and two sons.

WILLIAM E. GROVE, M.D.

Dr. William E. Grove, 63, died January 8, 1978, in Wichita. He was born August 23, 1914, in Newton.

Dr. Grove received his medical education at Northwestern University and served with the Army Medical Corps prior to returning to Newton, where he was associated with the Axtell Clinic.

Survivors include his wife, a daughter, and a son. A memorial fund has been established with Axtell Christian Hospital.

JOSEPH W. PARKER, JR., M.D.

Dr. Joseph Warren Parker, Jr., 76, died January 17, 1978, in Emporia. He was born November 28, 1901, in San Antonio, Texas.

Dr. Parker was graduated from the University of Kansas School of Medicine in 1945. He practiced in Kansas City and Burlington prior to coming to Emporia State University as director of the department of health in 1963. He retired in 1969.

Survivors include his wife and a daughter. Memorial contributions may be made to the Greater University Fund at the University of Kansas at Lawrence.

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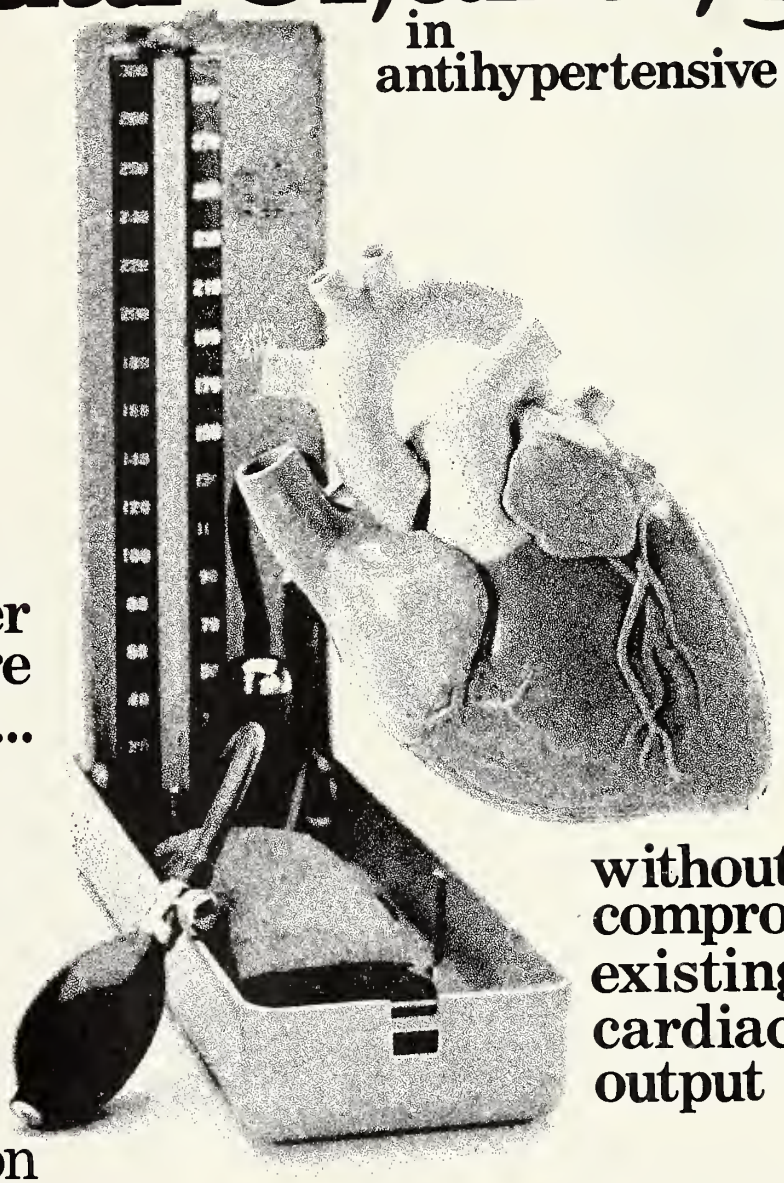
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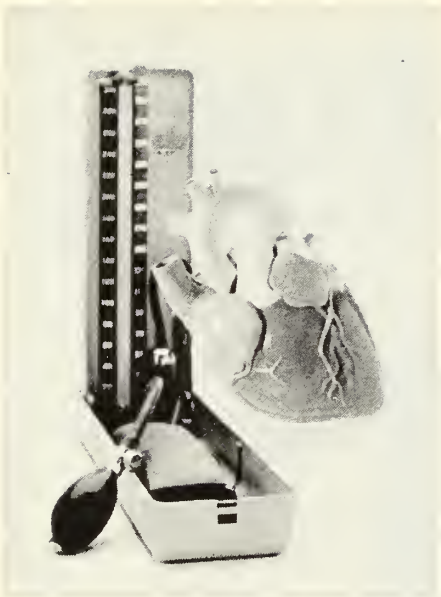
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Warnings: It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. Read this section carefully to understand these reactions.

With prolonged methyldopa therapy, 10% to 20% of patients develop a positive direct Coombs test, usually between 6 and 12 months of therapy. Lowest incidence is at daily dosage of 1 g or less. This on rare occasions may be associated with hemolytic anemia, which could lead to potentially fatal complications. One cannot predict which patients with a positive direct Coombs test may develop hemolytic anemia. Prior existence or development of a positive direct Coombs test is not in itself a contraindication to use of methyldopa. If a positive Coombs test develops during methyldopa therapy, determine whether hemolytic anemia exists and whether the positive Coombs test may be a problem. For example, in addition to a positive direct Coombs test there is less often a positive indirect Coombs test which may interfere with cross matching of blood.

At the start of methyldopa therapy, it is desirable to do a blood count (hematocrit, hemoglobin, or red cell count) for a baseline or to establish whether there is anemia. Periodic blood counts should be done during therapy to detect hemolytic anemia. It may be useful to do a direct Coombs test before therapy and at 6 and 12 months after the start of therapy. If Coombs-positive hemolytic anemia occurs, the cause may be methyldopa and the drug should be discontinued. Usually the anemia remits promptly. If not, corticosteroids may be given and other causes of anemia should be considered. If the hemolytic anemia is related to methyldopa, the drug should not be reinstituted. When methyldopa causes Coombs positivity alone or with hemolytic anemia, the red cell is usually coated with gamma globulin of the IgG (gamma G) class only. The positive Coombs test may not revert to normal until weeks to months after methyldopa is stopped.

Should the need for transfusion arise in a patient receiving methyldopa, both a direct and an indirect Coombs test should be performed on his blood. In the absence of hemolytic anemia, usually only the direct Coombs test will be positive. A positive direct Coombs test alone will not interfere with typing or cross matching. If the indirect Coombs test is also positive, problems may arise in the major cross match and the assistance of a hematologist or transfusion expert will be needed.

Fever has occurred within first 3 weeks of therapy, occasionally with eosinophilia or abnormalities in liver function tests, such as serum alkaline phosphatase, serum transaminases (SGOT, SGPT), bilirubin, cephalin cholesterol flocculation, prothrombin time, and bromsulphalein retention. Jaundice, with or without fever, may occur, with onset usually in the first 2 to 3 months of therapy. In some patients the findings are consistent with those of cholestasis. Rarely fatal hepatic necrosis has been reported. These hepatic changes may represent hypersensitivity reactions; periodic determination of hepatic function should be done particularly during the first 6 to 12 weeks of therapy or whenever an unexplained fever occurs. If fever and abnormalities in liver function tests or jaundice appear, stop therapy with methyldopa. If caused by methyldopa, the temperature and abnormalities in liver function characteristically have reverted to normal when the drug was discontinued. Methyldopa should not be reinstituted in such patients.

Rarely, a reversible reduction of the white blood cell count with primary effect on granulocytes has been seen. Reversible thrombocytopenia has occurred rarely. When used with other antihypertensive drugs, potentiation of antihypertensive effect may occur. Patients should be followed carefully to detect side reactions or unusual manifestations of drug idiosyncrasy.

Pregnancy and Nursing: Use of any drug in women who are or may become pregnant or intend to nurse requires that anticipated benefits be weighed against possible risks; possibility of fetal injury or injury to a nursing infant cannot be excluded. Methyldopa crosses the placental barrier, appears in cord blood, and appears in breast milk.

Precautions: Should be used with caution in patients with history of previous liver disease or dysfunction (see Warnings). May interfere with measurement of: urinary uric acid by the phosphotungstate method, serum creatinine by the alkaline picrate method, and SGOT by colorimetric methods. Since methyldopa causes fluorescence in urine samples at the same wavelengths as catecholamines, falsely high levels of urinary catecholamines may be reported. This will interfere with the diagnosis of pheochromocytoma. It is important to recognize this phenomenon before a patient with a possible pheochromocytoma is subjected to surgery. Methyldopa is not recommended for patients with pheochromocytoma. Urine exposed to air after voiding may darken because of breakdown of methyldopa or its metabolites.

Stop drug if involuntary choreoathetotic movements occur in patients with severe bilateral cerebrovascular disease. Patients may require reduced doses of anesthetics; hypotension occurring during anesthesia usually can be controlled with vasopressors. Hypertension has recurred after dialysis in patients on methyldopa because the drug is removed by this procedure.

Adverse Reactions: *Central nervous system:* Sedation, headache, asthenia or weakness, usually early and transient; dizziness, lightheadedness, symptoms of cerebrovascular insufficiency, paresthesias, parkinsonism, Bell's palsy, decreased mental acuity, involuntary choreoathetotic movements; psychic disturbances, including nightmares and reversible mild psychoses or depression.

Cardiovascular: Bradycardia, aggravation of angina pectoris. Orthostatic hypotension (decrease daily dosage). Edema (and weight gain) usually relieved by use of a diuretic. (Discontinue methyldopa if edema progresses or signs of heart failure appear.)

Gastrointestinal: Nausea, vomiting, distention, constipation, flatus, diarrhea, mild dryness of mouth, sore or "black" tongue, pancreatitis, sialadenitis.

Hepatic: Abnormal liver function tests, jaundice, liver disorders.

Hematologic: Positive Coombs test, hemolytic anemia. Leukopenia, granulocytopenia, thrombocytopenia. Positive tests for antinuclear antibody, LE cells, and rheumatoid factor.

Allergic: Drug-related fever, lupus-like syndrome, myocarditis.

Other: Nasal stuffiness, rise in BUN, breast enlargement, gynecomastia, lactation, impotence, decreased libido, dermatologic reactions including eczema and lichenoid eruptions, mild arthralgia, myalgia.

Note: Initial adult dosage should be limited to 500 mg daily when given with antihypertensives other than thiazides. Tolerance may occur, usually between second and third months of therapy; increased dosage or adding a diuretic frequently restores effective control. Patients with impaired renal function may respond to smaller doses. Syncope in older patients may be related to increased sensitivity and advanced arteriosclerotic vascular disease; this may be avoided by lower doses.

How Supplied: Tablets, containing 125 mg methyldopa each, in bottles of 100; Tablets, containing 250 mg methyldopa each, in single-unit packages of 100 and bottles of 100 and 1000; Tablets, containing 500 mg methyldopa each, in single-unit packages of 100 and bottles of 100 and 500.

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"Angel dust" is susceptible to "ion trapping" and that is good news to overdose victims of phencyclidine (PCP).

"Ion trapping" is a treatment technique described in "Phencyclidine Overdose: An Emergency Concept of Management," in a recent issue of *JACEP,* the medical journal for emergency medicine. The authors, Regine Aronow, M.D., Alan Done, M.D. and Joseph N. Miceli, Ph.D., developed the treatment to deal with the rising incidence of "angel dust" overdoses seen at The Children's Hospital of Michigan in Detroit.

Phencyclidine is found on the street in a variety of pill forms, capsules, dust, and occasionally as a liquid. It is sniffed, smoked, or swallowed. Often, it is represented to buyers as mescaline, cocaine, amphetamine, or LSD. Victims of overdose may suffer loss of tactile sensation, coma, convulsions, and respiratory failure.

Chronic users often arrive in the emergency department with psychiatric problems including acute anxiety, severe depression, and paranoid psychosis. With few references in the literature on the management of PCP, there has been little guidance available for physicians faced with a victim of PCP overdose.

The illicit use of PCP, developed in the late 1950s as an anesthetic for nonhuman primates, is fast becoming a nationwide problem. The National Institute of Drug Abuse estimates that as much as 30 per cent of all admissions to mental hospitals may be PCP-related. In Michigan alone, over 360 deaths were caused by PCP overdose in the past three years.

Dr. Aronow's treatment removes the drug from the victim's body as fast as possible by washing out the stomach and increasing the acidity of the urine which facilitates "ion trapping." In ion trapping, the acid medium forced into the patient's stomach through a tube is absorbed, and attracts the PCP ions already in the body's system like a magnet. Because acid is not reabsorbed into the body, the trapped PCP is flushed out of the body through the urine.

Dr. Aronow uses ammonium chloride and ascorbic acid, dissolved in saline as the acid medium. The rate at which the patient recovers is directly related to the amount of PCP taken from the body.

Once a patient regains consciousness, ascorbic acid is continued throughout the hospital stay. The patient is also encouraged to drink several glasses of cranberry juice a day. The juice has a high acid content, and continues the acidification program.

The treatment also involves support measures and close monitoring of vital signs. While the victim should be in a quiet, low-light environment, "talking down" is of no use in handling these patients.

In the group of patients studied, this approach to treatment was successful in all cases, including one who had been on respiratory assistance for nine days before the treatment was begun. Patients recovering from extreme overdose show a steady pattern of continuing improvement, unlike the uneven progress shown by the patients who received only supportive care.

HYPNOSIS GAINS APPROVAL

Hypnosis, like biofeedback, is gaining new popularity in the United States as a means of helping some people learn to deal with their health problems, says a report in a recent issue of *JAMA.*

Hypnosis has been used particularly to help patients deal with pain resulting from arthritis, cancer, or other chronic or life-threatening diseases. Hypnotherapy is being used for some psychosomatic illnesses, for reduction of nausea resulting from drug treatments in cancer patients, and for other problems, such as preoperative apprehension, nail biting, tension headaches, obesity, insomnia, and smoking, the *JAMA* report says.

The staff of the *JAMA Medical News* section presents in-depth study of the many uses of hypnosis as an adjunct to therapy for a variety of human ills. Some research indicates that for some disorders biofeedback and hypnosis are equally effective and can lead to the same changes in body processes.

Hypnosis is all around us, the report points out. An Oregon physician says: "In my opinion every physician is using hypnosis every day to varying degrees, and every patient goes into spontaneous hypnosis several times during the course of treatment."

A California psychiatrist points out that, "After a period of time, many patients' symptoms and physiological problems simply become conditioned habits, with the patients maintaining behavior they have learned."

When there is no physical reason for the symptoms, the physician might consider the possibility that the patient has learned to maintain the symptoms as an attention-getting device, as self-punishment because of guilt feelings, or as a means of meeting other psychological needs. Hypnotherapy can help these individuals overcome their symptoms.

Disease is not "cured" by hypnosis. It merely relieves symptoms, such as pain or nausea, and it still is vitally important to establish a proper diagnosis and treatment of the disease itself, in addition to using hypnosis, the report emphasizes.

About 5 per cent of the population apparently are not hypnotizable, about 20 per cent can go into deep trances, and the rest can go into light or medium trances, often becoming more hypnotizable with practice.

Hypnosis is more effective as treatment in dealing with

problems of physical origin than with problems of psychiatric origin, says a Harvard psychiatrist.

AMNESTY INTERNATIONAL

The Medical Advisory Board of Amnesty International, the human rights organization working for prisoners of conscience, conducted an international medical seminar on "Violations of Human Rights: Torture and the Medical Profession," in Athens on March 10 and 11, 1978.

The purposes of the seminar were, first, to report to and consult with the medical profession about the progress that has been made during the last three years with regard to the medical work against torture, including codes of medical ethics and medical research; and second, to further the implementation of these results and to encourage the medical profession to participate more in the different aspects of medical work against torture—specifically, to encourage the formation of Amnesty International medical groups in various countries. (Such medical groups already exist in Denmark, France, Greece, Holland, Sweden, and the U.S.A. Besides these groups, Amnesty International national sections maintain many contacts with medical organizations and individual professionals.)

It was especially fitting to hold the seminar in Athens, for Greece was the first country in which Amnesty International doctors systematically examined torture victims for the purposes of medical research against torture. The first results of this study were published by Amnesty International in 1977 in a booklet entitled *Evidence of Torture: Studies by the Amnesty International Danish Medical Group.*

With Greek doctors already interested in the problems of research and treatment in this field, the seminar provided an oportunity to discuss the medical detection and effects of torture as well as the need for treatment, rehabilitation, and compensation of torture victims in a context where these topics have immediate relevance.

Those who desire more information about the seminar or Amnesty International's medical work against torture are urged to write to: Amnesty International U.S.A., Western Regional Office, 3618 Sacramento, San Francisco, California 94118.

BIORHYTHM VALIDITY TEST

Biorhythm has been subjected to the test of scientific analysis and has been found wanting, says a report in *Archives of General Psychiatry.*

Biorhythm is a theory that purports to identify periods of increased individual susceptibility to accident or misfortune on the basis of recurring biological cycles. We have good days and bad days, and these days can be predetermined by applying the mathematical theory of biorhythm, proponents say.

A research group representing Johns Hopkins University School of Medicine, the Baltimore City Hospitals, the Maryland Medical-Legal Foundation, Inc., and the University of Maryland School of Medicine gathered data from 205 carefully investigated highway crashes.

Of the crashes, 135 were fatal, 70 nonfatal. In each, drivers were clearly at fault. The researchers then computed specific points in drivers' biorhythm cycles at which the accidents occurred. The observed frequencies of accidents occurring during so-called critical and minus periods were then compared with the frequencies to be expected on a chance basis alone.

Conclusion: "The results provided no evidence for a relationship between purported biorhythm cycles and accident likelihood."

The researchers add: "We don't consider this result either a cause for elation or an occasion for a pseudoscientific diatribe on the foibles and follies of the lay public. The theory had a certain wistful appeal; the fact that it was examined and found wanting cannot, therefore, be attributed to any negative bias that somehow crept into the tabulation and analysis of the data."

The biorhythm theory has the effect of the self-fulfilling prophecy, they point out. If you think you will have a good day, you just might have one. The same with a bad day. Chance and coincidence may be magnified by expectations.

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Respiratory Circulatory Dynamics

(Continued from page 124)

When bronchospasm is relieved and airway clearance effected, the weaning process is usually rapid. Bronchopneumonia with significant production of purulent sputum necessitates full ventilation, frequent suctioning, or bronchoscopy. Under these circumstances, mucus plugging is likely to increase if spontaneous ventilatory excursion is marginal.

Advantages of Intermittent Mandatory Ventilation

For the patient who can breathe but who may suffer from progressive fatigue, support can be more comfortably provided by an occasional assisted or controlled breath from a mechanical ventilator. This should be administered in a manner least likely to intrude upon the patient's spontaneous efforts. Cardiovascular depression is minimized, since mean intrathoracic pressure is less than with continuous ventilation. Also, circulatory adaptation to spontaneous breathing is facilitated. If brainstem response to CO_2 challenge is appropriate, swings in $PaCO_2$ due to hyperventilation are avoided. A support system is thus provided which interferes least with the patient's normal functions. Finally, the rhythmic employment of the respiratory musculature is retained, reducing the tendency to ventilator dependence, and to motor incoordination during the weaning process.

IMV requires close, expert surveillance. If continuous care of high quality is available, the patient needs only enough ventilator support to assure adequate gas exchange. If marked changes in compliance or resistance occur, if the patient is easily exhausted and cannot sustain the work of breathing or if apneic intervals are likely to develop (as in patients with head injury), the risks of intermittent delivery systems may be prohibitive, and continuous ventilation becomes essential.

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Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

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Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

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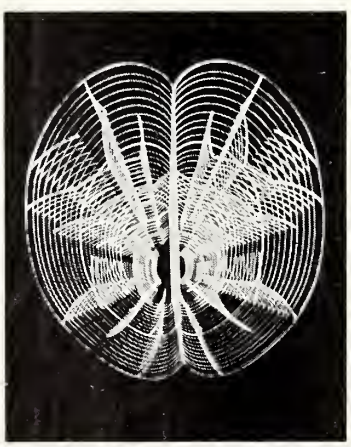
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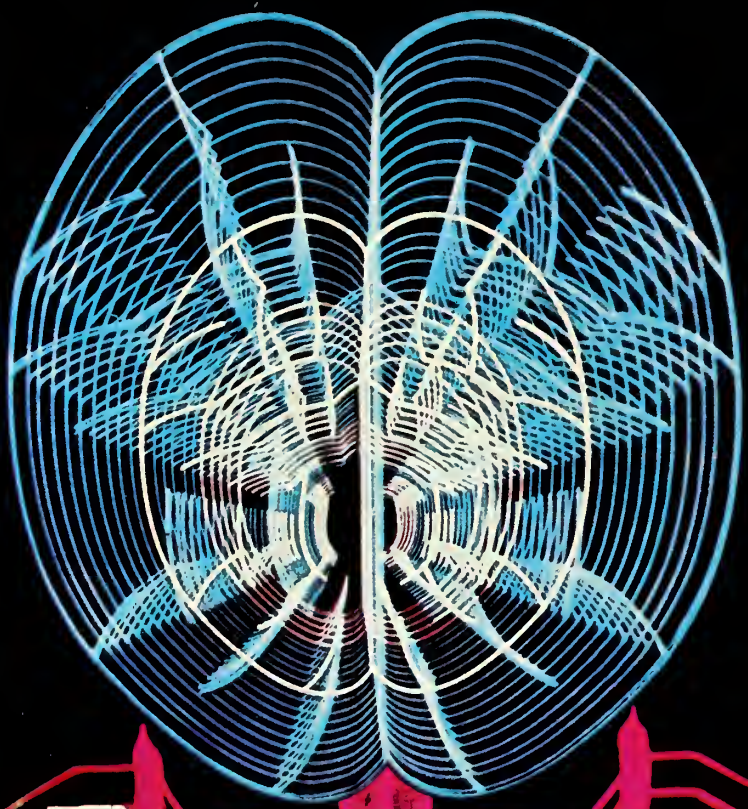
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APRIL
1978

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The JOURNAL of the KANSAS MEDICAL SOCIETY

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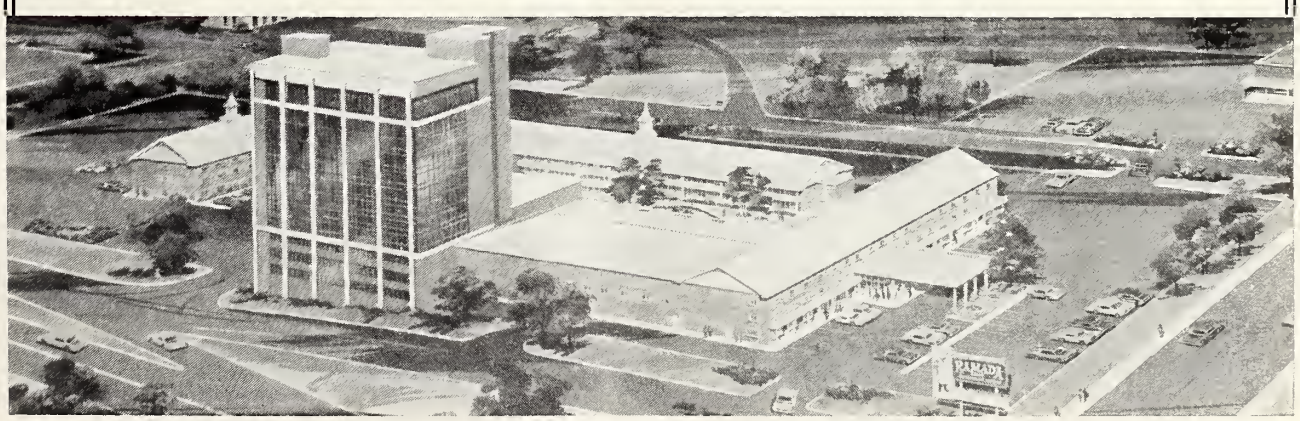
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Sunday, May 7–House of Delegates First Session, 9:00 AM–
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Monday, May 8–Sports Day, Topeka Country Club

Tuesday, May 9–Scientific Session,
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Wednesday, May 10–House of Delegates Second Session,
Ramada Inn Convention Center, 8:30 AM



Kansas Medical Assistants Society

38th Annual Convention

April 28-30, 1978, Downtown Ramada Inn, Topeka

Friday, April 28

3:00 Registration
Governor's Club

3:00 Executive Council Meeting
Parlor D

4:30 Leadership Seminars
Presidents Parlor D
Treasurers Room 201
Education/Certification Parlor B
Constitution/Bylaws Parlor A
Membership Parlor C

8:00 "The Long Branch Saloon"
Grand Ballroom
Courtesy Mumms Medical Supply, Inc.

Saturday, April 29

7:00 Self-Assessment Test
Parlor B

7:30 Registration, Coffee & Rolls
Governor's Club
Courtesy Berlin-Wheeler, Inc.

7:30 Past Presidents' Breakfast
Parlor D

9:00 General Session
Lower Lounge
Virginia Wells, Topeka, Speaker
Call to Order
Mary Klinge, President
Kansas Medical Assistants Society
Welcome
Robert E. Roeder, M.D., President
Shawnee County Medical Society
Greetings
Mary Jane Schmidt, President
Shawnee County Medical Assistants Society

12:00 CMA Luncheon—Regency Ballroom

12:00 Chapter Presidents' Luncheon—Parlor D

1:30 Medical Legislation
William R. Roy, M.D., J.D., Topeka

2:30 Epilepsy
Joseph M. Stein, M.D., Topeka

4:00 Aeronautical Medicine
William J. Reals, M.D., Wichita
OR
How to Handle the Difficult Patient
Dean T. Collins, M.D., Topeka

7:30 Inaugural Banquet
Grand Ballroom
Greetings
John D. Huff, M.D., President
The Kansas Medical Society
Master of Ceremonies
Ole R. Cram, Jr., M.D., Larned
Installation of 1978-1979 Officers
Entertainment

Sunday, April 30

7:45 Buffet Breakfast
Grand Ballroom

8:00 Executive Council Meeting
Parlor B

9:00 General Session
Lower Lounge
Mary Klinge, Presiding

9:30 How to Survive Parenthood
Alan D. Megibow, M.D., Topeka

10:45 Medical Practice in Smalltown USA
Emerson D. Yoder, M.D., Denton

12:00 Luncheon
Grand Ballroom
Presentation of Gavel
Introduction of 1979 Officers
Invitation to 1979 Convention

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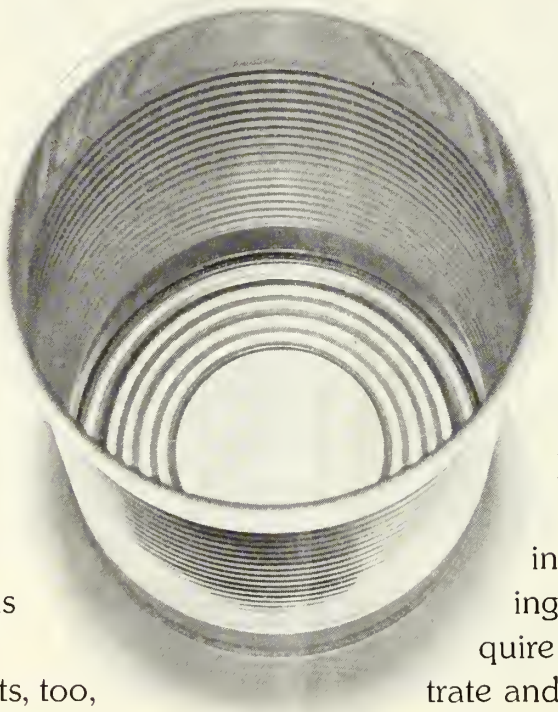
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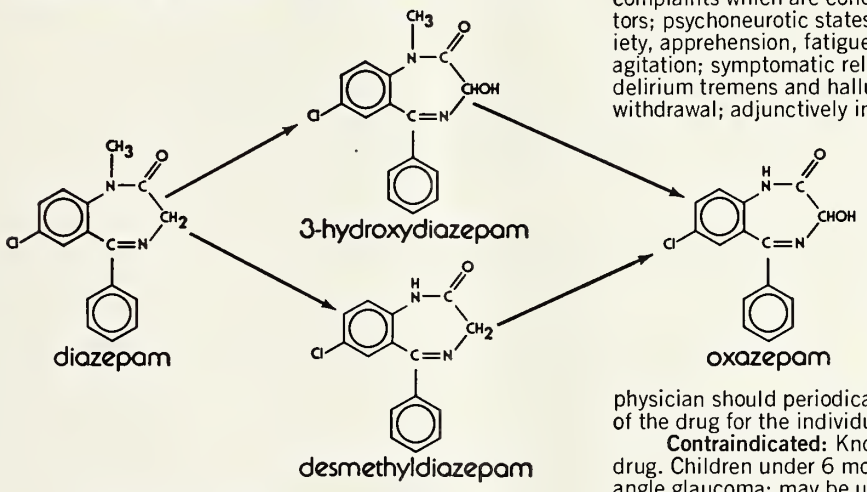
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The effectiveness of Valium (diazepam) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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House of Delegates

Ramada Inn Convention Center

SUNDAY–MAY 7

7:30 Registration of Delegates

9:00 First Session, Regency Ballroom II

WEDNESDAY–MAY 10

7:30 Registration of Delegates

8:30 Second Session, Grand Ballroom

Council Meeting and Luncheon at Conclusion of House of Delegates, Regency Foyer West

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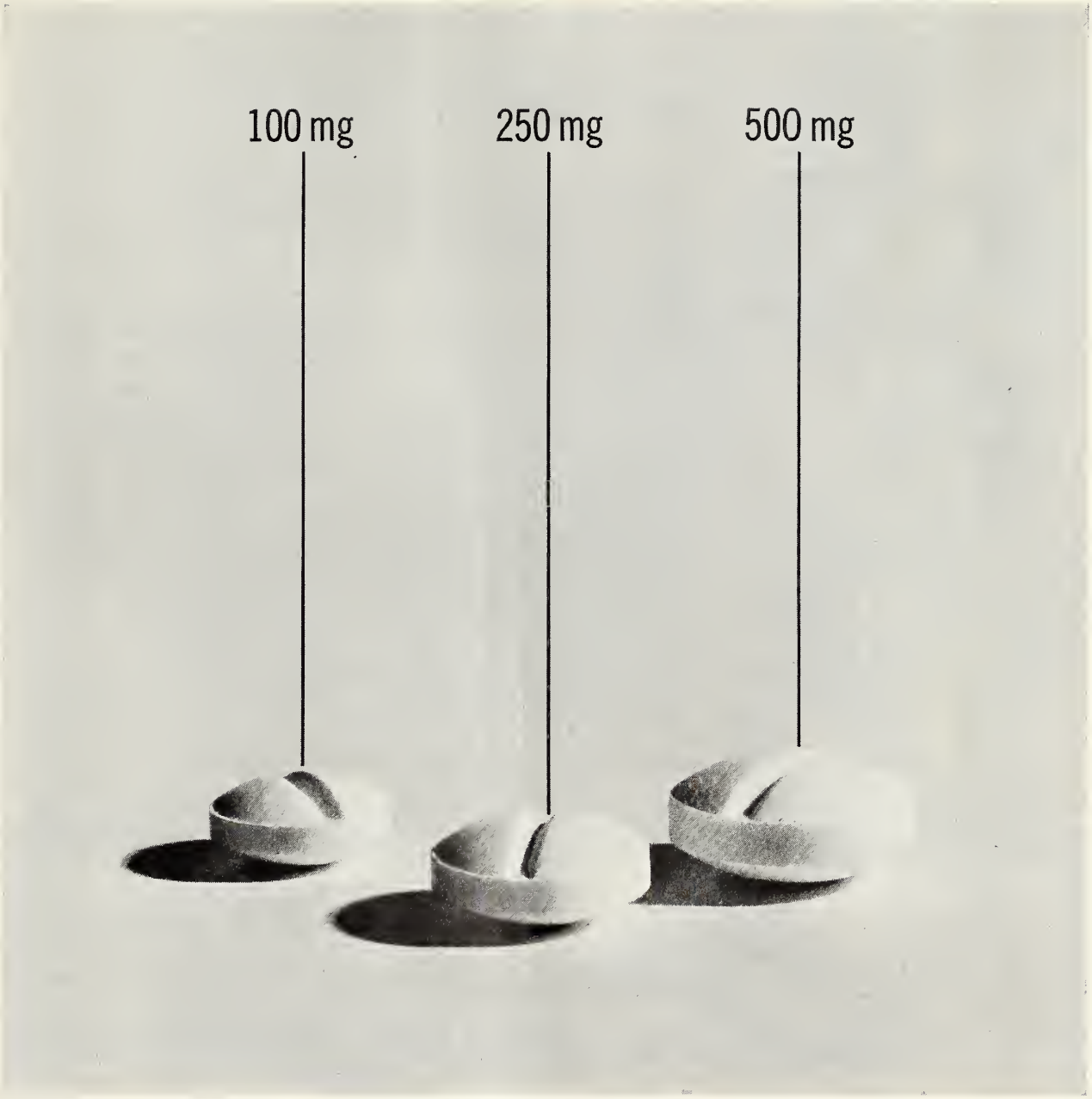
Reference Committees

SUNDAY, MAY 7–2:00 P.M.

Committee A–Regency Ballroom II

MONDAY, MAY 8–9:00 A.M.

Committee B–Regency Ballroom II



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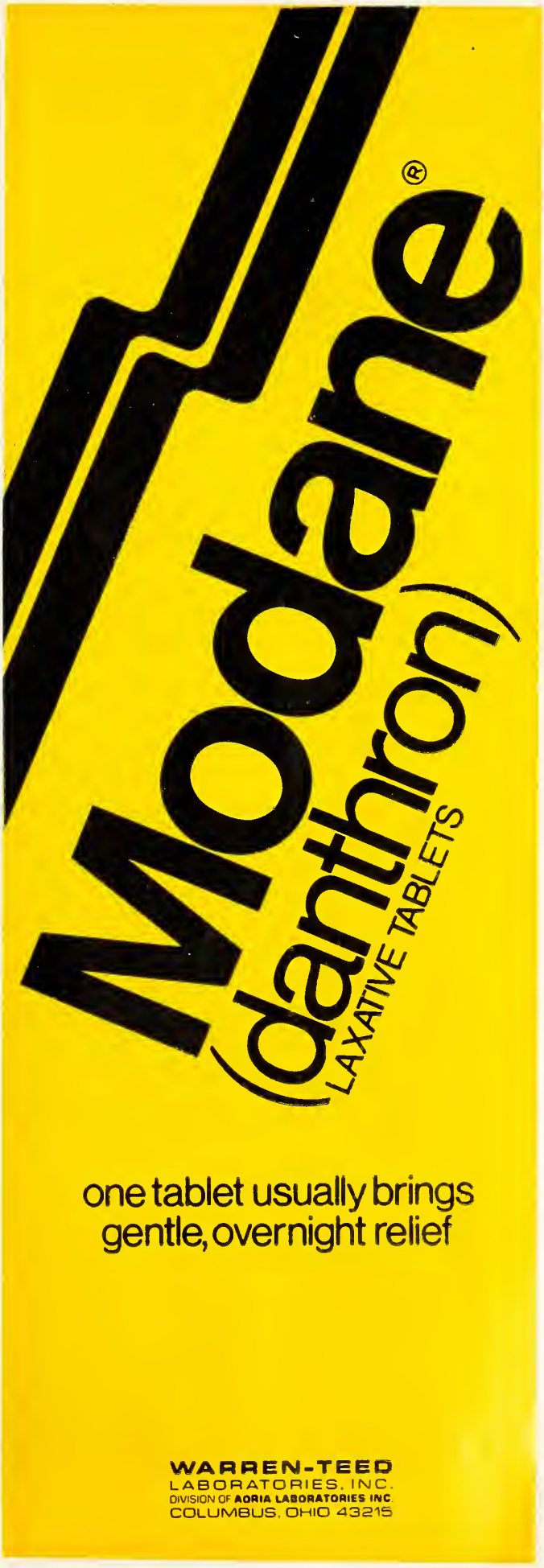
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The Kansas Medical Society Auxiliary

53rd Annual Convention, Ramada Inn Convention Center, Topeka

SUNDAY, MAY 7

11:00 Registration—Reservations
Parlor B

11:00 Hospitality and AMA-ERF Store
Parlor B
The ERF Store and Hospitality Room will remain open throughout the convention except during the special events.

1:00 Pre-Convention Board of Directors Meeting
Parlor B
Mrs. A. Dean Burnett, Halstead, President
The Kansas Medical Society Auxiliary
Presiding

6:00 Dinner-Theatre
Topeka Civic Theatre
Warehouse-on-the-Landing
534½ No. Kansas Ave. (map will be provided)
Buffet dinner served 6:00-7:15
Curtain 8:00
Capacity limit: 191. Reservations accepted at KMS or through the Auxiliary Reservation Chairman (Mrs. Robert F. Woods), on a first-come, first-serve basis.

MONDAY, MAY 8

7:30 "Early Bird Breakfast"
Regency Foyer
Michael Johnson, D.D.S., Chairman
Kansas House Public Health and Welfare Committee
Both KMS and KMSA members are urged attend. Sports Day participants are most welcome!

8:00 Registration—Hospitality—AMA-ERF Store
Parlor B

9:00 General Session Workshop
Lower Lounge South
Mrs. A. Dean Burnett, Presiding
Mrs. Chester L. Young, President
AMA Auxiliary
Mrs. Young will give the keynote address. Workshops on AMA-ERF, Membership, and Legislation will be conducted. All are cordially invited to this special session.

11:15 Ecology Day Lunch and Tour (Bus boarding from 6th St. entrance)
and
11:45 Two groups will tour Meade Gardens and Home; eat a delicious box luncheon; visit the Topeka Zoo Rain Forest; and enjoy punch and cookies while hearing about solar energy at the beautiful country home of Dr. and Mrs. Chet Lessenden. Reservations must be made in advance. Tickets include all costs: bus, luncheon, admission, and everything! Buses boarding, Ramada Inn
The event will honor Mrs. Chester Young and our State Officers.

6:30 KMS Sports Day Reception and Dinner

6:30 Non-Sports Social Hour and Dinner
This requested get-together is for those not involved in the Sports Day program, who would enjoy meeting friends for a pleasant dinner together. Dinner served at 7:30. B.Y.O.L. Reservations, please.

TUESDAY, MAY 9

7:30 Past State Presidents' Breakfast
Parlor A

8:30 Registration—Hospitality—AMA-ERF Store
Parlor B

9:00 General Session
Lower Lounge South
Mrs. A. Dean Burnett, Presiding
All delegates, members, friends are most welcome. Come and be a part of this special meeting.

12:30 Transportation to Luncheon

1:00 Punch Bowl and Luncheon
Shawnee Country Club
Mrs. A. Dean Burnett, Presiding
Honored will be John D. Huff, M.D., President of the Kansas Medical Society, and the KMS Advisory Committee on Auxiliary. Newly elected officers will be installed.

5:30 K.U. Medical Alumni Reception

7:00 KMS President's Banquet

WEDNESDAY, MAY 10

9:30 REGISTRATION—HOSPITALITY—AMA-ERF STORE
Parlor B

9:30 BREAKFAST
Parlor B

10:00 POST-CONVENTION BOARD MEETING
Mrs. William H. Crouch, Presiding
All new board members, county presidents and presidents-elect are urged to attend.

11:30 ADJOURNMENT

* * *

Pre-Registration Is Essential

Because of the limitations of time and space, your reservation must be received by May 4. Please be specific as to the events and the number of tickets desired. Include your check, payable to SHAWNEE COUNTY CONVENTION FUND, and address the reservations to: *Mrs. Robert Woods, Reservations Chairman, 2042 Birchwood, Topeka, KS 66604.*

Deadline for DINNER-THEATRE (Sunday, May 7) is APRIL 21. Please send your ticket order early. There are only 191 places available, and the first to come in will receive the tickets!

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The Three C's of Psychiatric Nursing: Conceptualization, Competency, Compassion—April 28-29, 1978. The role of the psychiatric nurse in the treatment setting will be examined.

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For further information please contact June Housholder, Division of Continuing Education, The Menninger Foundation, P.O. Box 829, Topeka, Kansas 66601. 913/234-9566, ext. 3685.



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DINNER MUSIC-THEATRE

Topeka Civic Theatre
Warehouse-on-the Landing
Sunday, May 7, 1978

THE SECRET AFFAIRS OF MARGARET WILD—
a comedy by Pulitzer Prize Winner Paul Zindel

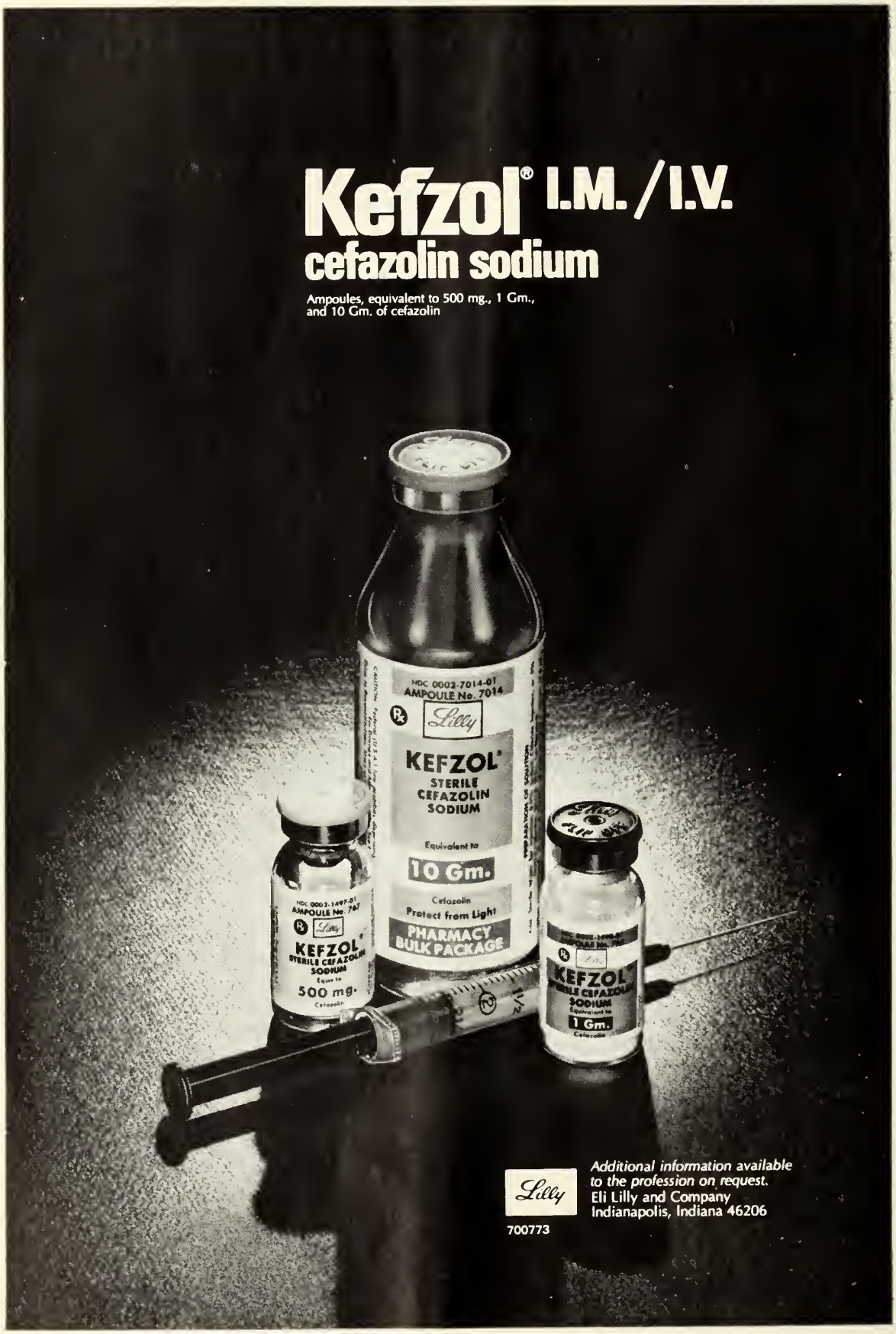
Buffet Dinner served 6:00-7:15
Curtain time 8:00



Reservations will be honored on a first-come, first-serve basis and must be made by April 21. Theatre capacity is 191, with most tables seating four persons. A limited number of tables for 2 and for 6 are available. There is one table for 3. Requests for persons to be seated in groups will be honored when possible. PLEASE LIST THOSE IN YOUR PARTY, INCLUDE THE TOTAL AMOUNT, AND ATTACH TO RESERVATION FORM. (TCT is a Class A private club, therefore alcoholic beverages including wine are available. Set-ups, wine corkage and tips are extra.)

Please indicate type of reservation you wish by checking below:

For Non-TCT Members:	**Cost Per**	**No.**	**Total**
Dinner and Show	@ $9.50	_____	$_____
Alcoholic beverage charge per person	$2.00	_____	$_____
Non-alcoholic beverages only (do not include liquor charge)			
Administrative charge per person	$.50	_____	$_____
For TCT Members Only			
Season Membership to TCT applicable to this play (no ticket charge)			
Dinner only (for all but sustaining members)	$7.00	_____	$_____
Existing liquor pool will be used			
Not drinking alcoholic beverages			
Administrative charge per person	$.50	_____	$_____
Total enclosed for your party			$


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Welcome to Topeka

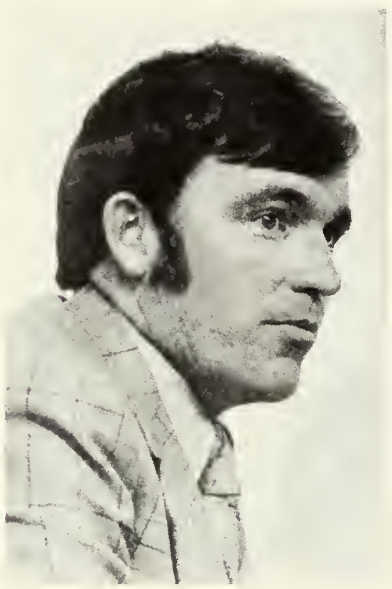
The Physicians and Auxiliary of the Shawnee County Medical Society are most honored and pleased to serve as host of the Annual Meeting of the Kansas Medical Society for '78.

We cordially welcome Councilors, Delegates, Members, Spouses, and Families to Topeka.

Please call on our county staff to assist you in maintaining a pleasant visit.

Robert E. Roeder, M.D., President
The Shawnee County Medical Society, Inc.

Distinguished Speakers



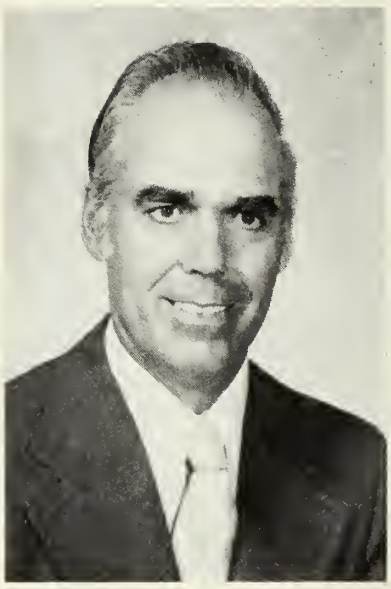
JOHN CARLIN

A native Kansan, House Speaker Carlin graduated from Kansas State University with a degree in Dairy Science. He presently operates the fourth generation Carlin Dairy Farm near Smolan.

Representative Carlin was first elected in 1970, to serve the 73rd District. The district consists of rural Saline County, the first precinct in the city of Salina, and Ellsworth County. He has served as House Minority Leader since 1975, and was elected Speaker of the House for the 1977-78 sessions.

Speaker Carlin holds leadership responsibilities in the following committees: Finance Council, Legislative Coordinating Council, and Legislative Budget Committee. In addition, he sits on the following committees: Agriculture and Livestock, Water Resources, Ways and Means, and Education. He also holds the appointment to the Supreme Court to the Judiciary Study Advisory Council, and serves as the Task Force Chairman for the National Conference of State Legislators' (NCSL) Committee on Rural Development.

Speaker Carlin will address the question of the Legislative Approach to Improving Medical Care.



ROSS O. DOYEN

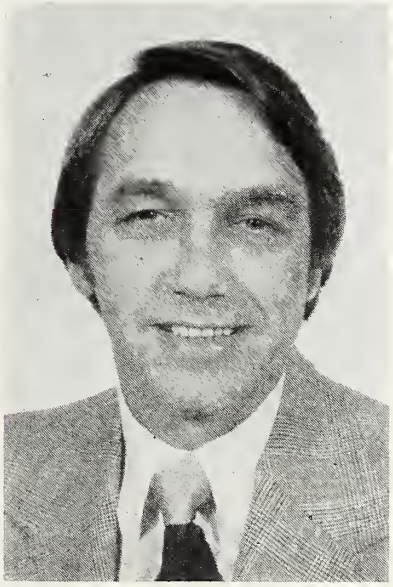
President of the Kansas Senate, Ross O. Doyen is a native of Cloud County. A graduate of Kansas State University, he was elected to the Kansas House of Representatives in 1958, where he served five terms. He was subsequently elected to the Senate, and served as chairman of the powerful Senate Ways and Means Committee.

As President of the Senate, Mr. Doyen alternates as chairman or vice-chairman of the Legislative Coordinating Council, the Interstate Cooperation Committee, and the Legislative Budget Committee. He is also a member of the State Finance Council and the State Building Advisory Committee.

Mr. Doyen, a farmer/rancher, is a member of the Executive Committees of the Council of State Governments and the Midwestern Conference of the Council of State Governments, and has served as chairman of the Agriculture Committee of the Midwestern Conference. He also serves on the Executive Committee, and the Governmental Operations Task Force of the Intergovernmental Relations Committee, of the National Conference of State Legislatures.

Mr. Doyen will discuss "The Legislative Approach to Improving Medical Care."

Distinguished Speakers



MICHAEL G. JOHNSON, D.D.S.

One of the most knowledgeable members of Kansas Legislature on health matters is Dr. Michael G. Johnson, a practicing dentist in Abilene. He obtained his DDS degree from the University of Missouri (Kansas City) Dental School. He has been practicing dentistry in Abilene for the past 15 years.

Dr. Johnson is a second-term Representative of the 67th District. He is Chairman of House Public Health and Welfare Committee. He is a member of the Governmental Organization Committee. He is also a member of the state-wide Health Coordinating Council.

A member of the Kansas State Dental Association and the American Dental Association, Dr. Johnson will be the featured speaker at the "Early Bird" Breakfast, on Monday morning .



OTHA W. LINTON

Mr. Linton is Director of Governmental Relations of the American College of Radiology, Washington Office. His areas of responsibility cover Executive Branch agencies with health programs involving radiation and radiology services, and include management of several contracts for health care projects. The responsibility also involves daily contact with legislative consultants in preparation of testimony.

A Master of Science in Journalism (University of Wisconsin '54), Mr. Linton is a free-lance writer, dealing primarily with health topics. He has been published by *JAMA, Journal of the Canadian Medical Association, Medical World News, Postgraduate Medicine, Medical Tribune,* and many others.

The topic of Mr. Linton's presentation is, "Medicine on the Washington Scene."

DAVID BLUMENTHAL

(no photograph available)

Mr. David Blumenthal is a member of Professional Staff, Subcommittee on Health, Senate Committee on Human Resources, Washington, D. C.



JOSEPH C. MEEK, JR., M.D.

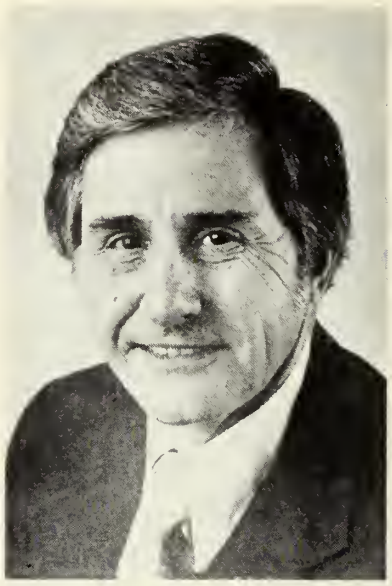
A native Kansan, Dr. Meek received his MD degree from the University of Kansas School of Medicine, in 1957. His internship and residency were served in the San Diego County General Hospital and at KUMC. He was a research assistant in space medicine at the U. S. Naval School of Aviation Medicine, Pensacola, Florida. Dr. Meek also served as fellow and trainee in endocrinology at Scripps Clinic and Research Foundation, La Jolla, California.

In his association with the University of Kansas Medical Center, Dr. Meek holds the following positions: Professor of Medicine; Director, Office of Rural Health Affairs; Director, Outreach Administration, KU College of Health Sciences; Associate Director, Division of Continuing Education.

A Fellow of the American College of Physicians, Dr. Meek is a Diplomate of the American Board of Internal Medicine and the Subspecialty Committee on Endocrinology and Metabolism. He has been a member of the Kansas Medical Society since 1966.

A prolific writer, Dr. Meek has co-authored an extensive number of scientific research papers.

Dr. Meek's presentation will deal with the "University of Kansas Health Care Outreach Program."



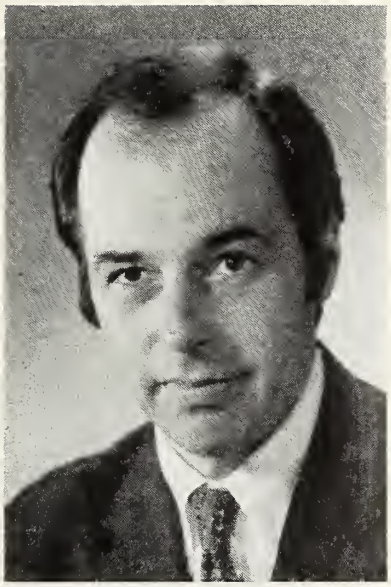
GEORGE M. PENN, M.D.

Born in Orange, New Jersey, Dr. Penn received his M.D. degree from the University of Texas Medical Branch, Galveston, in 1958. His internship and residency were served in John Sealy Hospital, Galveston. Additional residency in psychiatry was served in Austin State Hospital, Austin, Texas. After devoting a number of years to private practice of psychiatry in Lubbock, Texas, Dr. Penn joined C. F. Menninger Memorial Hospital, Topeka, where he is section chief.

Dr. Penn is Chairman of the Kansas Medical Society Committee on Physician Performance. He currently serves on the Professional Advisory Council of Kansas Medical Health Association, and is a member of the Board of Directors of National Council on Alcoholism.

A member of the Kansas Medical Society since 1969, Dr. Penn is certified by American Board of Psychiatry and Neurology, and he is a Fellow of the American College of Physicians. His special interests lie in professional and research aspects of hospital treatment, group and family treatment, alcoholism and other chemical dependence, with emphasis on physician impairment.

Dr. Penn will deliver his address at the General Luncheon, on Tuesday, May 9. He will describe the program initiated by the Physician Performance Committee of KMS, whose primary effort is directed at recognizing and aiding the impaired physicians of Kansas.



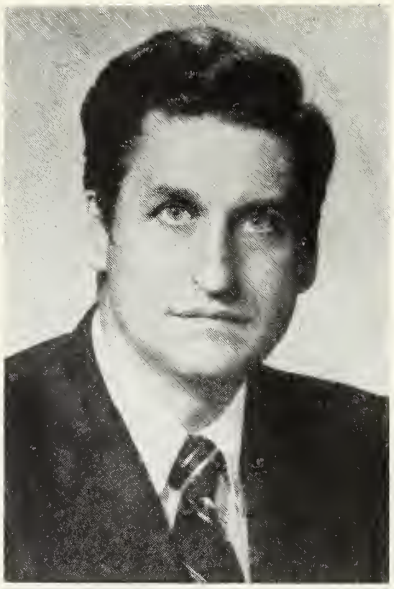
ALLEN LEE PINKERSON, M.D.

Born in Providence, Rhode Island, Dr. Pinkerson received his M.D. degree from Boston University School of Medicine. His internship and residency were served in Montefiore Hospital, Bronx, New York.

Currently he holds the following appointments: Medical Advisor, Bureau of Health Planning and Resources Development, Public Health Service; Instructor, Department of Medicine, and Lecturer, Department of Physiology and Biophysics, Georgetown University School of Medicine, Washington, D. C.

Dr. Pinkerson is certified by the American Board of Medical Examiners and American Board of Internal Medicine; he is a fellow of AHA Council on Circulation. He was the recipient of the honorary Angiology Annual Award for Outstanding Research in 1967.

A prolific writer, Dr. Pinkerson will discuss the National Guidelines for Health Planning.



WILLIAM ROBERT ROY, M.D., J.D.

Dr. Roy is currently Director of Medical Education and Professional Services at St. Francis Hospital, Topeka, and Executive Director of the Shawnee County Medical Foundation. He is a graduate of Northwestern Medical School and Washburn University Law School. His internship and residency were served in Evanston Hospital Association, Evanston, Illinois, and Herman Kiefer and City of Detroit Receiving Hospital. He is a Diplomate of the American Board of Obstetrics and Gynecology.

In private practice of medicine from 1955 until 1970, specializing in obstetrics and gynecology, Dr. Roy was elected to Congress in 1970, where he served four years. During that time, Dr. Roy co-authored extensive legislation, including the Emergency Medical Services Systems Act and the Health Maintenance Organization Act. He is presently a member of the Advisory Committee on National Health Insurance.

Dr. Roy is one of 330 members of the Institute of Medicine of the National Academy of Science, and is a member of the Committee on Health Care Needs, U. S. Chamber of Commerce.

A member of the Kansas Medical Society since 1955, Dr. Roy was elected Vice Speaker of its House of Delegates in 1970.

The topic of Dr. Roy's presentation is, "Overview of Federal Reimbursement Proposals."



JAMES W. WILSON, M.D.

Born near Ripley, Oklahoma, Dr. Wilson volunteered for flight training with the U. S. Army Air Corps in 1944, and later served in the Korean Conflict. He began his medical studies at the University of Oklahoma School of Medicine in 1954, where he obtained his MD degree in 1958. After serving his internship and residency, Dr. Wilson entered family practice in Oklahoma.

In 1968, Dr. Wilson was accepted to the resident staff in pathology at the University of Kansas School of Medicine. He received his board certification from the American Board of Pathology in clinical and anatomic pathology in 1970.

Dr. Wilson was on the staff of the University of Missouri School of Medicine, Kansas City, as Associate Professor of Pathology and Director of Clinical Pathology at General Hospital. He concurrently served as Director of Nuclear Medicine at the Veterans Administration Hospital, Kansas City. Dr. Wilson subsequently practiced medicine in Coffeyville. He joined the Kansas Medical Society in 1972.

He became Director of Health for the Kansas Department of Health and Environment in July 1977. In his address, Dr. Wilson will discuss the work of his Department.



MRS. CHESTER L. YOUNG

Kansas City, Kansas

President, American Medical Association Auxiliary

Mrs. Young, who has been active at many levels of Auxiliary work, took office as President of the American Medical Association Auxiliary at the 1977 annual convention in San Francisco. Her activities with the national Auxiliary have included serving in various capacities on the Board of Directors and as chairman of many committees. An Auxiliary member for more than 25 years, Mrs. Young served in a number of capacities at the state level, and was elected president of the KMS Auxiliary in 1961.

A "true Midwesterner," Lela May Carter Young was born in DeKalb County, Missouri, and graduated from Trinity Lutheran Hospital School of Nursing in Kansas City. She was married to the late Chester L. Young, M.D., a surgeon.

Mrs. Young has resumed her nursing career on a part time basis. She is active in civic affairs in many areas of community, political, and church work.

The Kansas Medical Society Auxiliary will honor Mrs. Young with the ECOLOGY DAY LUNCH AND TOUR, on Monday, May 8.

PRESIDENT'S BANQUET
MAY 9, 1978



The Croatian heritage—its culture, its music—is flourishing in Kansas City through the talents of the **St. John C. C. Tamburitzans.**

Organized with 34 boys and girls in 1966, the overall program now includes more than 100 young people who play Yugoslavian instruments—brac, prim, and bugarija—and perform the dances of Yugoslavia and of several neighboring countries. Students begin training during fourth or fifth grade, and advance to the performing company when they reach eighth grade.

Under the direction of conductor Don Lipovac, the performing group of 40-50 boys and girls present an annual concert, and perform locally and in other cities.

The Tamburitzans have performed with the Philharmonic Orchestra; they have also been guests of Yugoslavia on two occasions, traveling about the country to present numerous programs. To date, the group has produced six records.

Hosts for the Meeting

Shawnee County Physicians Arranging 1978 Session

GENERAL CHAIRMAN—Richard Meidinger, M.D.

PROGRAM COMMITTEE

Joseph E. McMullen, M.D.
Robert D. Porter, M.D.
William R. Roy, M.D.
Harry H. White, M.D.

SPORTS DAY

Louis Cohen, M.D.
Merle D. Morris, M.D.
John Runnels, M.D.
Millard C. Spencer, M.D.

Sunday, May 7, 1978

Ramada Inn Convention Center

7:00 KANSAS FOUNDATION FOR MEDICAL CARE BOARD OF DIRECTORS MEETING
Parlor D

7:30 REGISTRATION—TICKETS—INFORMATION
Registration Area

9:00 HOUSE OF DELEGATES—FIRST SESSION
Regency Ballroom II
Clair C. Conard, M.D., Dodge City, Speaker
John O. Yulich, M.D., Kansas City, Vice Speaker

10:00 EXHIBITS OPEN
Regency Ballroom I

11:00 ENT SECTION
Luncheon-Meeting
Parlor C

11:00 KANSAS OBSTETRICAL & GYNECOLOGICAL SOCIETY
South Lower Lounge

12:00 KANSAS SOCIETY OF ANESTHESIOLOGISTS
Luncheon-Meeting
Parlor D

12:30 KANSAS ALLERGY SOCIETY
Luncheon-Meeting
Room 201

12:30 DELEGATES' LUNCHEON
Regency Foyer

1:00 SECTION ON NUCLEAR MEDICINE
Oval Room

2:00 REFERENCE COMMITTEE A
Regency Ballroom II
Alex Scott, M.D., Junction City, *Chairman*
Kenneth L. Derrington, M.D., Shawnee Mission
Vernon W. Filley, M.D., Pratt
Harry H. White, M.D., Topeka
R. Burnley White, M.D., Winfield

4:00 **KaMPAC HOSPITALITY SUITE (Everyone Invited)**
Governor's Club

4:00 EXHIBITS CLOSE
Regency Ballroom I

6:00 DINNER MUSIC-THEATRE
Topeka Civic Theatre
Warehouse-on-the-Landing, 534½ N. Kansas Ave.

VISIT THE EXHIBITS—REGISTER FOR DRAWING

A COFFEE LOUNGE WILL BE OPEN IN THE EXHIBIT AREA FOR PORTIONS OF THE CONVENTION

TELEPHONE NUMBER 913-233-8981

Monday, May 8, 1978

Ramada Inn Convention Center

MORNING

7:30 Registration—Tickets—Information
Registration Area

7:30 "Early Bird" Breakfast
With the KMS Auxiliary
Regency Foyer
Representative Michael Johnson, Abilene
Chairman, House Public Health and Welfare Committee

8:30 Exhibits Open
Regency Ballroom I

9:00 Reference Committee B
Regency Ballroom II
Evan R. Williams, M.D., Dodge City, *Chairman*
Ernest P. Carreau, M.D., Wichita
Norman G. Marvin, M.D., Kansas City, KS
G. Rex Stone, M.D., Manhattan
James M. Stout, M.D., Hutchinson

11:00 Exhibits Close

VISIT THE EXHIBITS—REGISTER FOR DRAWING

A COFFEE LOUNGE WILL BE OPEN IN THE EXHIBIT AREA FOR PORTIONS OF THE CONVENTION

SPORTS DAY ACTIVITIES

Kansas Medical Society Golf, Skeet and Trap Association
Merle D. Morris, M.D., Topeka, President

8:00 Golfing—Topeka Country Club
2700 Buchanan (Tel.: 354-8350)

9:00 Tennis—Topeka Country Club

6:30 Cocktails
Topeka Country Club
2700 Buchanan

8:00 Dinner
Topeka Country Club

TELEPHONE NUMBER 913-233-8981

Tuesday,

Ramada Inn Convention Center

MORNING

7:30 Registration—Tickets—Information
Registration Area

7:30 Past President's Breakfast
Foyer I West

8:30 Exhibits Open
Regency Ballroom I

First General Session

Regency Ballroom II

Richard Meidinger, M.D., Topeka,
Moderator

8:00 Welcome
Robert E. Roeder, M.D., President
Shawnee County Medical Society
Response
John D. Huff, M.D., President
The Kansas Medical Society

8:15 Overview of Reimbursement Alternatives
William R. Roy, M.D., J.D., Topeka

9:00 Medicine on the Washington Scene
Otha W. Linton, Chevy Chase, Maryland

9:40 Intermission to View Exhibits

10:00 How Physicians Can Best Participate in and Affect the Shaping of National Health Policy
David Blumenthal

10:40 Federal Health Planning Guidelines
Alan Pinkerson, M.D.

11:20 Panel Discussion
David Blumenthal
Otha W. Linton
Alan Pinkerson, M.D.
William R. Roy, M.D., J.D.

12:00 General Luncheon
Kansas Program on the Impaired Physician
George M. Penn, M.D., Topeka, Chairman
. . .KMS Physician Performance Committee

May 9, 1978

Ramada Inn Convention Center

SECOND GENERAL SESSION

Robert D. Porter, M.D., Topeka, Moderator

1:10 Kansas State Department of Health and Environment
James W. Wilson, M.D., Director

1:15 Report From the Governor's Special Committee on Medical Care in Kansas
Frank A. Lowman, Hays

1:35 The Legislative Approach to Improving Medical Care
Senator Ross O. Doyen, R-Concordia

1:55 House Speaker John Carlin, *D-Smolan*

2:15 Intermission to View Exhibits

2:35 Future of Continuing Medical Education
William R. Roy, M.D., J.D., Topeka

2:55 The University of Kansas Health Care Outreach Program
Joseph C. Meek, Jr., M.D., Kansas City, Kansas

3:30 Panel Discussion—*Harry H. White, M.D., Moderator*
Ross O. Doyen
John Carlin
Frank A. Lowman
Joseph C. Meek, Jr., M.D.
William R. Roy, M.D., J.D.

5:00 Exhibits Close

As an organization accredited for continuing medical education, the Shawnee County Medical Foundation Certifies that this continuing medical education offering meets the criteria for 6 credit hours in category I of the Physician's Recognition Award of the American Medical Association.

TELEPHONE NUMBER 913-233-8981

Tuesday, May 9, 1978

Ramada Inn Convention Center

EVENING

The President's Annual Banquet

5:30 Reception for Physicians and Spouses
Regency Foyer
Sponsored by K.U. Medical Alumni Association

7:00 Dinner
Grand Ballroom

John D. Huff, M.D.,
Presiding

Introduction of Guests

Oath of Office to Incoming President

President and President-Elect



John D. Huff, M.D.
Kansas City, Kansas



Warren E. Meyer, M.D.
Wichita

KMS ANNUAL MEETING



Doctor and Mrs. John D. Huff extend a personal invitation to members of the Society and their guests



to attend the President's Banquet on Tuesday evening, May 9, 1978.

Tuesday, May 9, 1978
Grand Ballroom
Ramada Inn Convention Center, Topeka

Wednesday, May 10, 1978

Ramada Inn Convention Center

MORNING

7:30 Registration—Information

8:30 House of Delegates—Second Session
Grand Ballroom

1:00 Council Luncheon and Meeting
Foyer West

NECROLOGY REPORT

Following is a list of the members of the Kansas Medical Society whose deaths have been reported since the last meeting of the House of Delegates.

Name and Address	*Age*	*Date*
William W. Abrams, *Kansas City*	70	Oct. 20, 1977
Kenneth E. Conklin, *Abilene*	74	Sept. 22, 1977
J. O. Gilliland, *Herington*	68	Aug. 29, 1977
Leland L. Holbert, *Kansas City*	62	Nov. 10, 1977
Cecil C. Hunnicutt, *Sabetha*	73	Nov. 24, 1977
Victor R. Moorman, *Hutchinson*	61	Sept. 8, 1977
William J. Porter, *Pratt*	51	Aug. 17, 1977
Harcharan S. Sehdev, *Topeka*	41	July 7, 1977
Margaret L. Shiney, *Topeka*	50	Oct. 2, 1977
Francis M. Stone, *Fort Scott*	62	Oct. 29, 1977
Alonzo A. Towner, *Varadale, WA*	67	Sept. 23, 1977

THE KANSAS SOCIETY OF PATHOLOGISTS

invites the pathologists and urologists of Kansas to the SEMINAR ON UROLOGICAL PATHOLOGY and the SEMINAR ON QUALITY CONTROL IN THE LABORATORY, both to be held on Saturday, May 13, 1978, in Topeka. Category I credit will be allowed by the American Society of Clinical Pathologists.

Slides for the Urological Pathology Seminar will be available soon. For slides and information, please contact:

Antonio Huaman, M.D.
1700 West 7th St.
Topeka, KS 66606

Exhibits

The exhibits, located in Regency Ballroom I, Ramada Inn, will be open Sunday, 10:00 a.m. to 4:00 p.m.; Monday, 8:30 a.m. to 11:00 a.m., and Tuesday, 8:30 a.m. to 5:00 p.m.

Register at the exhibit booths for drawings to be held at the dinners on Monday and Tuesday evenings.

Booth No.	
1	Boehringer Ingelheim, Ltd. Elmsford, New York
2	Munns Medical Supply Company, Inc. Topeka, Kansas
3	Cooper Laboratories, Inc. Parsippany, New Jersey
4	The Medical Protective Company Fort Wayne, Indiana
5	Sandoz Pharmaceuticals East Hanover, New Jersey
6	Wyeth Laboratories Philadelphia, Pennsylvania
7	Kansas Beef Council Topeka, Kansas
8	Smith Kline and French Laboratories Philadelphia, Pennsylvania
9	Mallinckrodt, Inc. St. Louis, Missouri
11	Bristol Laboratories Syracuse, New York
12	United States Air Force Medical Recruiting Kansas City, Missouri
13	Johnson and Johnson Dermatological Division New Brunswick, New Jersey
17	Kansas Brace & Limb, Inc. Wichita, Kansas
18	Modern Office Methods Control-O-Fax Topeka, Kansas
19	United States Army Medical Department Washington, D.C.
20	United Professional Investors, Inc. Shawnee Mission, Kansas
21	Searle Laboratories Chicago, Illinois
22	Vita Mix Corporation Cleveland, Ohio
27	Reed and Carnrick Pharmaceuticals Kenilworth, New Jersey
28	William H. Rorer, Inc. Fort Washington, Pennsylvania
29	Flint Laboratories Deerfield, Illinois
30	Washington National Insurance Company Topeka, Kansas

The Kansas Medical Society is grateful for the convention program grants received from:

A. H. Robins Company
Richmond, Virginia

Merck Sharp & Dohme
Shawnee Mission, Kansas

A COFFEE LOUNGE WILL BE OPEN IN THE ASSEMBLY AREA THROUGHOUT THE CONVENTION

Councilor Reports

Activities in the Council Districts of Kansas

DISTRICT 1

During this past year, Council District 1 has been relatively quiet, and some encouraging news has been forthcoming on several different fronts.

First, some of our towns short of physicians (Hiawatha, for example) have been delighted to acquire a complement of new physicians. Several other cities in our area have available to them prospective new medical talent, which should go far toward relieving the physician shortages long present in northeast Kansas.

Second, the progress in communicating with our state legislators has resulted in some positive influence in the legislative process within the State of Kansas. This progress, by helping to shape decisions "up front," as it were, is always much more satisfying than attempts to shape legislative action by reacting to it after it has been presented to the legislature itself.

Lastly, we look forward to the same type of productive, constructive effort in communicating with those of our political acquaintances who are running for office during this upcoming election year, secure in the knowledge that failure to involve oneself on a personal basis in the political process leaves in its wake a vacuum, the results of which may well be somewhat unpleasant for medicine in its efforts to serve Kansas.

ROGER D. WARREN, M.D., *Councilor*

DISTRICT 2

The main issue confronting physicians in Council District 2 during the past year remained political. As some leaders will recall, the local HSA is made up of counties on both sides of the Missouri-Kansas state line. The group serving this function, known as MASHA, submitted a very comprehensive, and some thought unrealistic, plan for consideration by the Wyandotte County Medical Society. As nearly as we could determine none of our suggestions had any impact.

Membership in the District remained about the same this year following several years of steady growth. There is some interest in developing a paging system to be run by the county medical society. Other than these two small items, for the Second District, the status remained *quo* during the past year.

ROBERT P. HUDSON, M.D., *Councilor*

DISTRICT 3

District 3 of the Kansas Medical Society represents the entire County of Johnson. The metropolitan areas in the District have experienced remarkable expansion in the past year, as is the case in other metropolitan areas in the state. The Johnson County Medical Society has added 18 members in the past year. Expansion of the physical facilities of the two existing hospitals in the county has been accomplished. The opening of a third hospital is scheduled for sometime this calendar year. The problems confronting the private practice of medicine continue to escalate. The growing "cancer" of regional government continues to pose unending problems in the consideration of health facilities and doctors in the county. Contending with MASHA and the despotism of Federal Regionalism inhibits the exercise of the free market. The private practice of medicine contends with a federally subsidized HMO in an adjoining county, and a similar contrivance is planned to be opened in this county within the calendar year. The physicians in this District of the Kansas Medical Society look forward with some apprehension to the development of a possible schism in the county society, developing from a yet-to-be-organized staff for a new hospital and its HMO in the area. Medical society districts and counties in Kansas bordering other states have continuing problems with the tentacles of regionalism, and this problem is most prevalent in the metropolitan area of Kansas City. A resolution concerning itself with the impact of Federal Regionalism on the private practice of medicine, as well as the state-at-large, has been under consideration by the Council of your medical society and will be re-introduced at the House of Delegates meeting for implementation. In my opinion, the efforts to hybridize three eastern counties in Kansas is not only undesirable, but is clearly unconstitutional.

LEWIS G. ALLEN, M.D., *Councilor*

DISTRICT 5

Council District 5 has been fairly stable. Several new physicians have joined the district, expanding the specialties.

We had a fine dinner meeting with our president, John D. Huff, M.D., late in November. He was joined

by Jerry Slaughter, the Executive Secretary. Numerous medical subjects and legislative problems were explained.

RICHARD H. O'DONNELL, M.D., *Councilor*

DISTRICT 6

Shawnee County Medical surrounds of Shawnee County have changed considerably during the past year. We have seen an active building and expansion program in all of our primary care medical facilities. As yet, the community has not benefitted from additional beds, but should see expanded medical facilities and new major services offered continuously throughout 1978. Within the past year we have seen the opening of the Capital Region Radiotherapy Center at St. Francis Hospital and Medical Center. Memorial Hospital has opened an acute psychiatric care unit and also has opened a unit specializing in the care of the ophthalmologic patients. Stormont-Vail Regional Medical Center has continued to operate a successful open heart program to serve Topeka and the surrounding region. During these expansion programs and new service developments we have found it necessary to learn again to deal with the various planning agencies. The new HSA has made its appearance and is struggling to identify itself as a valid and feasible agency of the federal government, as well as a feasible representative of local medical systems.

This year, as last year, one of the major efforts of the medical community was directed toward expansion of medical education. The Shawnee County Medical Foundation continues to be the guiding body for medical education and has found it possible to distribute residents and training programs quite equitably about the community. This past year the Foundation studied in detail the possibility of developing a Family Practice Residency in Topeka. After extensive evaluation of the programs, we found it more feasible locally to expand our interests in primary care, *i.e.,* general internal medicine, pediatrics, and obstetrics and gynecology. The necessity of developing a free standing educational structure and outpatient service made it financially impossible to proceed with the development of the family practice program at this time. It seemed to many of us that the physical guidelines required in the process of programmed development for family practice residency which now prevails places the entire program in jeopardy on both a local and national basis.

During 1977, the Foundation provided residency rotations for 50 residents in internal medicine, obstetrics and gynecology, pediatrics, family practice and general surgery (thoracic experience). The number of residents served in each specialty was as follows:

Internal Medicine (K.U. 21, Menninger School of Psychiatry 10)	31
Pediatrics	5
Obstetrics and Gynecology	7
Family Practice	2
General Surgery	5

Additionally, the Foundation continued its AMA-approved Grand Rounds and Clinical Seminars programs. These programs included Combined Grand Rounds, Surgery Grand Rounds, Pediatric Grand Rounds, the Clinical Pathology Conferences, Oncology Conferences, Neuroradiology Conferences and seminars on Chronic Renal Failure, Biofeedback, Clinical Blood Banking, Radiation Therapy, and Clinical Radiology. The foundation also cosponsored the Kansas Chapter of the American College of Surgeons. During the year, a total of 288 individual physicians participated, earning a total of 5,626 hours of AMA Category I Credit. We of Shawnee County have found the Foundation to be a flexible and excellent mechanism to expand local educational experiences and to expand our relationship with the University of Kansas School of Medicine.

We are looking forward to serving as your host during the coming Kansas Medical Society convention. Bring your spouse, your sports apparel, your good humor and your good weather, and join us in May.

MILLARD C. SPENCER, M.D., *Councilor*

DISTRICT 7

Council District 7 is comprised of six counties (Lyon, Morris, Waubaunsee, Osage, Coffee, Chase) most of whose physicians are members of the Flint Hills District Medical Society. With the exception of physicians in Waubaunsee County, all are chartered members of the Flint Hills District Medical Society. William H. Walker, M.D. of Eskridge, Waubaunsee County, is a welcome voluntary member of the Flint Hills Society. Monthly meetings, mostly at the Emporia Country Club, have been well attended. Good programs have been arranged by program chairman, Stephen Knecht, M.D. State president, John D. Huff, M.D., updated our knowledge of state society activities at the November meeting. At that time, Jerry Slaughter also made a good slide presentation challenging us to become more concerned and involved with the affairs of government and medicine.

Two new full-time physicians have been added to our ranks since the last Kansas Medical Society state meeting, namely: David Edwards, M.D., orthopedic surgeon, and John Harvey, M.D., obstetrician-gynecologist. For reasons of health, our only otorhinolaryngologist, E. L. Gann, M.D., retired. We are greatly in need of a replacement in his field. Well-liked psychiatrists, Deja Suthi-

kant, M.D. and Wm. R. LeVine, M.D., left our district for new fields of endeavor, the latter having joined KUMC-Wichita faculty. Since their departure, Robert Kieth, M.D., of the Menninger Clinic staff, is serving Newman Hospital and the Emporia based Mental Health Center of East Central Kansas on a part-time basis. Two Wichita Clinic Dermatologists, Drs. Stanley L. Cooper and John E. Schlicher, see patients in Emporia on alternate Mondays at the Flint Hills Medical Building.

Significant additions to the physical plants of both Emporia hospitals are under construction.

The KUMC Easter Circuit course continues to provide monthly postgraduate education at the Newman Hospital School of Nursing Building.

There have been no noteworthy disciplinary problems in the past year.

MARVIN D. SNOWBARGER, M.D., *Councilor*

DISTRICT 9

Council District 9 met on January 19, for the annual meeting with the President of the Kansas Medical Society, Dr. John D. Huff. At that time, some of the legislative matters were discussed. The meeting was well attended.

The Saline County Medical Education Foundation has been organized during the past year and presently is providing some excellent accredited postgraduate medical education. The status of the extension of the proposed family practice residency is still uncertain at this time.

A number of new physicians have moved into our Council district during the past year. They are in Abilene: Gary W. Coleman, M.D. and J. Steven Schwarting, M.D.; in Salina: Richard R. Brummett, M.D., Ted L. Macy, M.D., and Ramon W. Schmidt, M.D.; in Beloit: John M. Loney, M.D. Two osteopathic physicians will be serving the Lincoln area under the National Health Service Administration beginning in July.

KERMIT G. WEDEL, M.D., *Councilor*

DISTRICT 10

Council District 10 physicians and their spouses met at the Prairie Dunes Country Club, Hutchinson, on February 22, 1977. Emerson Yoder, M.D. and Jerry Slaughter spoke to the group on matters relating to the Kansas Medical Society. We heard an update on March 28, 1978, from John D. Huff, M.D. and Jerry Slaughter.

The physicians of McPherson, Marion, and Harvey counties have each hosted the tri-county medical society meetings in their area during the year.

Mrs. Jackie (Dean A.) Burnett, of Halstead, has made District 10 very happy and proud of her service as President of KMS Auxiliary this past year.

District 10 physicians are looking forward to hosting the Kansas Medical Society Annual Meeting in the new Holidome, Hutchinson, in 1979.

HERBERT FRANSEN, M.D., *Councilor*

DISTRICT 11

The following is the report of Council District 11 for 1977-78:

January 1977: Dr. Charles Jackson accepted the gavel as incoming president from President Dr. Curtis Drevets. Dr. Jackson's major concerns were with the problem of health care cost containment and establishment of a program for getting new and young physician members involved in MSSC activities.

February 1977: The February program for 1977 was presented by John F. Budd, M.D., AMA President-Elect.

March 1977: The meeting in March was a joint meeting with the pharmacists with a panel discussion by four society physicians, led by Dr. William Swisher, member of the Kansas State Healing Arts Board, on how to deal with problems resulting from the use and misuse of drugs.

Our Legislative Committee, chaired by Dr. H. T. Gray, was very active during the annual session of the Legislature. An informal meeting hosting all of the legislators from Sedgwick County was held for the purpose of assisting them regarding any health related legislation.

April 1977: In April, Ed Joseph Carels, Ph.D., Assistant Vice-President of Blue Shield, spoke on the rising health care costs from Blue Shield's viewpoint.

May 1977: The annual business meeting of the membership was held with Dr. John J. Schlueter being named President-Elect for 1979, and Dr. Richard Cummings named President of MSSC as of January 1, 1978.

The society's Professional Investigation Committee, Board of Directors, and Executive Secretary spent a great deal of time during the past year on the drug abuse problem. Wichita, with one-fourth of the state's population, is a high risk, urban area and it was estimated that cost to the city and the county exceeded more than $6 million in 1974, because of drug-related problems.

The KUMC-Wichita Branch continues to grow and contribute to the needs of the region and the state. Forty-three family practice MDs have started practice in the past three years in 23 different counties. These phy-

sicians were trained at KUMC and the Wichita Branch.

The Emergency Medical Service of the city-county continues to operate and do an excellent job, thanks to considerable time and medical guidance by members of MSSC. Dr. Ernest McClellan is chairman of the advisory committee and a physician member of the Community Board of Health, which supports and guides EMS in a responsible manner attempting to keep it free from potential political entanglements.

MSSC Career Loan Fund, thanks to the support of physicians and medical auxiliary members, has $30,000 outstanding in loans and another $20,000 in available loans to qualifying medical students and paramedical students in training.

July 1977: The MSSC Insurance Review Committee, which has been active since 1977, ceased to review fees as requested by patients and third parties and was disbanded. This was the result of legal advice that medical societies should limit review to physicians' services, medical necessity, and appropriateness of medical care.

MSSC surveyed the 196 members who joined the society between 1972 and 1977 and obtained the following information:

	Per Cent
Attended medical schools in other states	45.9
Attended foreign medical schools	28.5
Graduated from KUMC	25.5
Received residency training:	
in other states	53.0
in Wichita	35.0
in Kansas City area	8.0
in foreign countries	3.0

MSSC Standing Committee on Health Planning is currently reviewing the 200-page HSA comprehensive health systems plan so that our representatives to the Southeast Kansas HSA and sub-area councils, Drs. Hugo Weber and H. T. Gray, can represent the health planning needs as perceived by the physicians of District 11.

Sedg-Pac, a separate and free-standing organization, has been formed in District 11 for the purpose of supporting local candidates for state legislature. Dr. Joe Robertson is chairman of Sedg-Pac.

Local Advisory Board to McConnell AFB has been established for the purpose of improving military and civilian relations in the medical field. Dr. Tom Kendall is the MSSC representative.

November 1977: Dr. William R. Roy discussed national health insurance and medical care as a political issue. MSSC Board and Blue Shield have agreed to conduct a one-year experimental program to determine cost savings on selected medical procedures when provided in physicians' offices, versus in hospital setting, either as in-patient or out-patient. The experiment is designed to determine whether higher physician reimbursement in office practice would affect hospital utilization pattern and thus result in cost savings.

December 1977: The December 1977 meeting was presented by KUMC-Wichita. Dr. D. Cramer Reed, Vice-Chancellor; Richard Welch, M.D., Dean; and Mike Worley, Director of Institution Research and Planning, KUMC, discussed cost and funding of medical education.

January 1978: Dr. John D. Huff, President of KMS, was the guest speaker at our regular society meeting.

The MSSC Investigative Committee, after considerable review, made specific recommendations to the Sedgwick County commissioners for changes at the E. B. Allen Memorial Hospital Holding Ward. These were designed to determine who would have authority over the unit and the type of medical and psychiatric care rendered.

MSSC was proud to have been the host for the 118th Annual Session of the Kansas Medical Society held in Wichita, on May 1-4, 1977.

IVAN E. RHODES, M.D., *Councilor*

DISTRICT 12

The component societies of Council District 12 have met regularly and on schedule, and have had scientific subjects presented of high caliber. A keen interest in medical society affairs has been apparent. Each society is preparing to evaluate the problems presented in resolutions in the upcoming spring meeting of the Kansas Medical Society.

Area hospitals have been progressing in acquisition of equipment that updates these modalities of the past and is, therefore, increasingly helpful to the area patients. In our area at the Harper District Hospital, a computer terminal has been placed through the University of Kansas School of Medicine-Wichita, and is available for use of the faculty and interested physicians in the area, as well as students there.

Specialty physicians are increasing their services locally so that, overall, the area of medical output is of higher quality than before. One paper emanating from this district has been published on the *Journal of the Kansas Medical Society* during this period of activity.

It is apparent that Council District 12 component societies have been progressively updated and active in the medical, and legislative affairs in which the Kansas Medical Society is involved.

P. J. ANTRIM, M.D., *Councilor*

DISTRICT 13

During the past year, the physicians of Central Kansas Medical Society have augmented their numbers by wel-

coming as new physicians to this area Dr. Thomas W. Hicklin, Smith Center, and Drs. Michael Wurm and Eric Musser, Phillipsburg.

Several preceptees were welcomed to our community during the past year.

The Central Kansas Medical Society was addressed by Dr. John D. Huff who, with his wife, visited Hays on September 22, 1977.

The High Plains Medical Education Consortium continues to sponsor accredited continuing medical education programs, and during this year was re-accredited for an additional two-year period. Increasing participation and a generally good reception are encouraging to the members of the Continuing Medical Education Committee.

During the course of this past year, the Radiology Department of Hadley Regional Medical Center has installed and are utilizing computer assisted nuclear cardiology unit. Also in 1977, the linear accelerator was installed in St. Anthony Hospital and was, in early 1978, put into active use.

The Hays Pathology Laboratory, serving the two Hays hospitals, moved into new quarters.

L. Wm. Halling, M.D., *Councilor*

DISTRICT 16

The Northwest Kansas Medical Society met with President John D. Huff, M.D., on December 1, 1977.

New physicians to the Northwest Kansas area are: Gerald R. Hassett, M.D., Colby—Radiology; Daniel R. Dunn, M.D., Colby—Family Practice; James Van Delden, M.D., Atwood—Family Practice; and Rosse E. Jewell, M.D., St. Francis—Family Practice.

Doctors from Quinter, St. Francis, Colby, Goodland, Hoxie, Oakley, Atwood and Oberlin are participating in the Kansas University Medical School Preceptorship Program.

In addition to the Preceptor Program, the Colby Clinic is now affiliated with the Family Practice Residency Program of St. Josephs Hospital, Wichita.

It is the hope of the medical society that these two programs will give the participants a greater understanding of practice in rural Kansas, and eventually alleviate the shortage of physicians in this part of the state.

Floyd L. Smith, M.D., *Councilor*

DISTRICT 17

The Southwest Kansas Medical Society held four meetings in 1977; two were scientific presentations and two informational type meetings with guest speakers. Dr. and Mrs. John D. Huff, our state President and wife, were guests at the fall meeting, when Dr. Huff gave us a summary of the state activities. Our December meeting was the annual election of officers for 1978. Richard Kalbac, M.D. was elected President; James Barnard, M.D., Vice-President; and Frank Eichhorn, M.D., Secretary-Treasurer.

We were quite optimistic about having a Family Practice Residency Program sponsored by KUMC and Dr. Edward Jenkins relocated to Garden City to head up this program. However, it was not approved for this year. Dr. Jenkins has opened his private practice of Family Medicine in Garden City, while he continues to work on the future approval of the Family Practice Residency Program. We continue to enjoy having a part in the training of medical students and residents. There have been 22 medical students from KU who have received part of their training in Garden City last year, as well as six medicine residents and four surgery residents. There were also four surgery residents from the University of Colorado School of Medicine who rotated through the program at St. Catherine's Hospital in Garden City.

Like all of rural Kansas, we continue to feel the shortage of general and family practitioners of medicine. The Garden City Medical Specialists have recently changed their name to Garden Medical Clinic, P.A. They have finished their building and have added a number of new occupants. Dr. Luther Fry moved his practice of ophthalmology from Dodge City to Garden City, and Drs. John Gilbert, orthopedic surgery; Stephen Meyers, pediatrics; and Michael Patterson, physical medicine, have joined the clinic group.

Family practice physicians and our projected residency program, along with water conservation and the agricultural strike, have dominated the medical-political scene and all will need solutions if Southwest Kansas is to continue to progress and develop in 1978.

Max E. Teare, M.D., *Councilor*

DISTRICT 18

District 18 has been fortunate to have an influx of new physicians in this area: one has set up private practice in Ottawa, and six physicians have come to Lawrence during the past year.

Ottawa has been upgrading their ambulance service which remains private, but there are plans to base them in the hospital in the future.

The new hospital in Lawrence is open and most of the growing pains of the new building have been solved. An item of considerable controversy has been the hospital's plan to build a physicians' office building when there are many office spaces presently available.

(Continued on page 180)

The Kansas Medical Society—1977-1978

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Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

Librax®

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium Br.

Please consult complete prescribing information, a summary of which follows:

Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

ROCHE

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A significant advantage of adjunctive

Librax®

Each capsule contains
5 mg chlordiazepoxide HCl and
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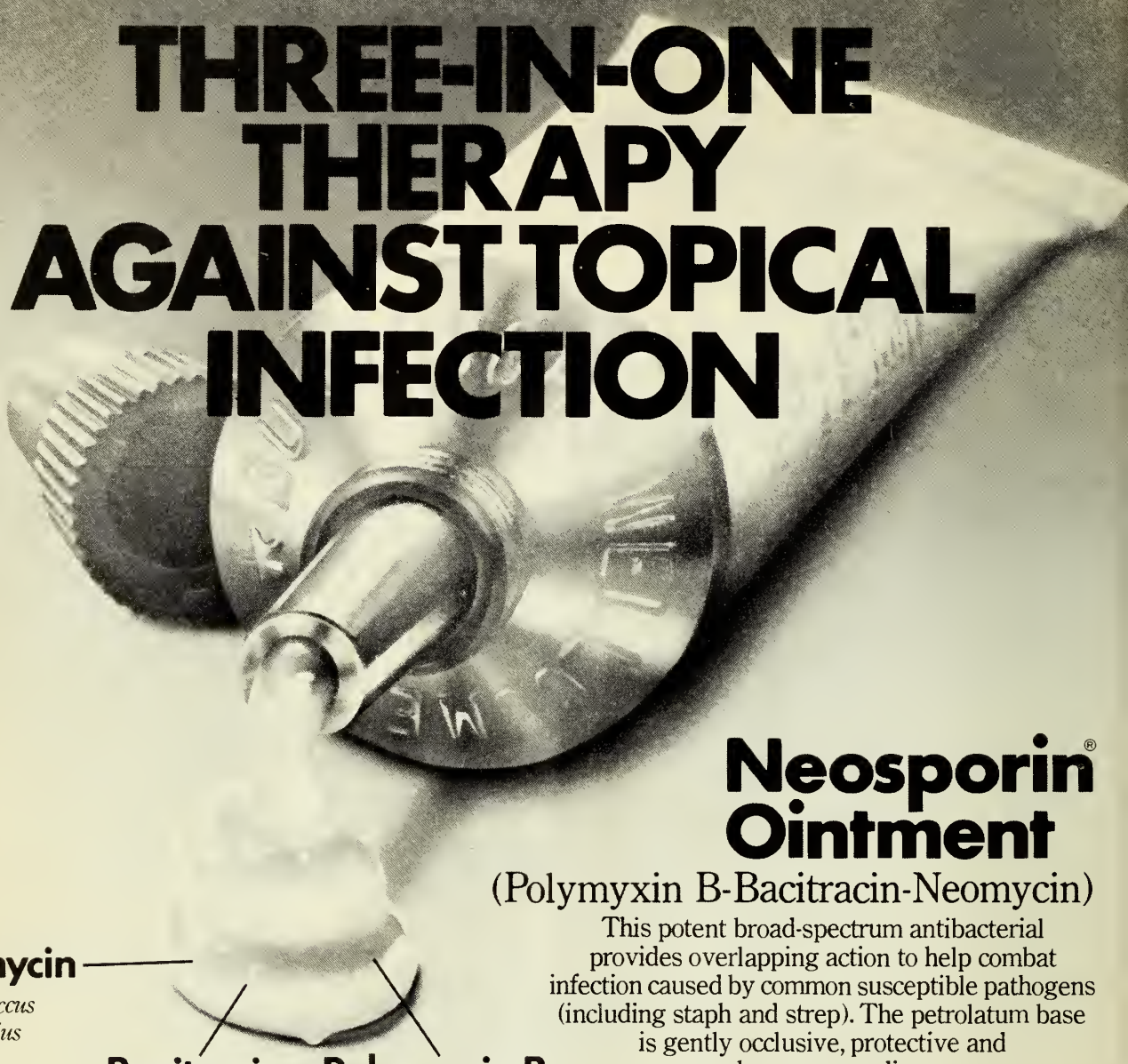
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*Librax has been evaluated as possibly effective for this indication.
Please see brief summary of prescribing information on preceding page.

ROCHE

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This potent broad-spectrum antibacterial provides overlapping action to help combat infection caused by common susceptible pathogens (including staph and strep). The petrolatum base is gently occlusive, protective and enhances spreading.

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In vitro overlapping antibacterial action of Neosporin® Ointment (polymyxin B-bacitracin-neomycin).

Wellcome

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

Neosporin® Ointment

(Polymyxin B-Bacitracin-Neomycin)

Each gram contains: Aerosporin® brand Polymyxin B Sulfate 5,000 units; zinc bacitracin 400 units; neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

TRIAMTERENE CONSERVES POTASSIUM WHILE HYDROCHLOROTHIAZIDE LOWERS BLOOD PRESSURE

DYAZIDE®

Each capsule contains 50 mg. of Dyrenium® (triamterene, SK&F Co.) and 25 mg. of hydrochlorothiazide.

MAKES SENSE



Before prescribing, see complete prescribing information in SK&F Co. literature or PDR. A brief summary follows:

* **Warning**
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** When the combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome. Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium sparing action of triamterene is warranted. (See Box Warning.) Routine use of diuretics in healthy pregnant women is inappropriate; they are indicated in pregnancy only when edema is due to pathological causes.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

Supplied: Bottles of 100 and 1000 capsules: Single Unit Packages of 100 (intended for institutional use only).

FOR LONG-TERM CONTROL OF HYPERTENSION.* SERUM K^+ AND BUN SHOULD BE CHECKED PERIODICALLY. (SEE WARNINGS SECTION.)

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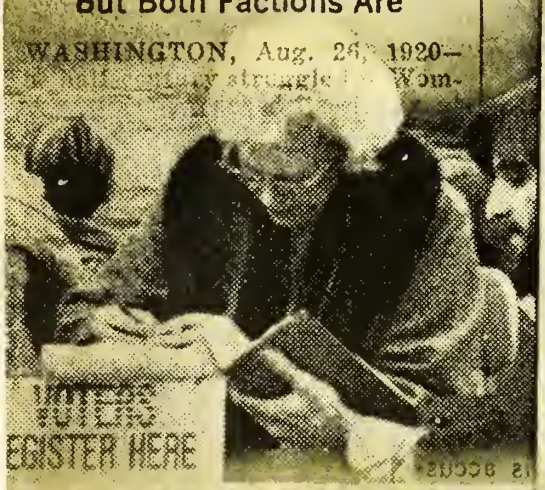
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MILITANTS VEXED AT PRIVACY.

Wanted Movies of Ceremony, But Both Factions Are

WASHINGTON, Aug. 26, 1920—



Social Security Bill Is Signed; Gives Pensions to Aged, Jobl

Roosevelt Approves Message Intended to Benefit 30,000, Persons When States Adopt Cooperating Laws–He Ca the Measure 'Cornerstone' of His Economic Program.

WASHINGTON, Aug. 14, 1
The Social Security Bill, prov
a broad program of unemploy
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Mr. Roosevelt cal[illegible] mea
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which is being [illegible]
me[illegible]s comple[illegible]

COM

SENATE APPROVES 18-YEAR OLD VOTE IN ALL ELECTIONS

Amendment to Constitution is Sent to House, Where Passage is Expected

WASHINGTON, March 10, 1971—The Senate approved today, 94 to 0, and sent to

TRUMAN CLOSES TED NATIONS CONFEREN ITH PLEA TO TRANSLATE CHARTER INTO DEEDS

NEW WORLD HOPE

President Hails 'Great Instrument of Peace,' Insists It Be Used

HISTORIC LANDMARK

Meeting Gives Standing Ovation as Executive Pictures Peace Gain

"If we fail to use it," he declared to the solemn final meeting of the delegates, 'we shall betray all of those who have died in order that we might meet here in freedom and safety to create it.'

"If we seek to use it selfishly—for the advantage of any one nation or any small group of nations—we shall be equally guilty of that betrayal."

Fervent Interpolation

The President, speaking in the auditorium of the War Memorial Opera House, built in memory of sons of the Golden Gate city who gave their lives in the first World War, in which he himself served, seemed to give unconscious expression to the solemn feeling of the occasion when, at the outset of his speech, he interpolated the words, half a hope, half a prayer:

"Oh, what a great day this can be in history!"

Just before the plenary session

IGNE

the Draf

Ends Nov

WASHINGTON, Jan. 27, 1973—"With the signing of the peace agreement in Paris today, and after receiving a report from the Secretary of the Army that



PATIENT PACKAGE INSERTS: A CONCEPT WHOSE TIME HAS COME?

The consumer's right to know is an irreversible and desirable trend of the Seventies. It extends, and properly, to a patient's right to know more about his or her prescription medications. One way, gaining favor, is through patient package inserts. Wisely-prepared and properly distributed when medically indicated, they could markedly improve patient knowledge and drug therapy—laudable goals by anyone's standards.

The PMA endorses these goals and will work with government, the health professions and consumers to achieve them.

The Advantages

The concept holds promise of benefits: better patient understanding of the product prescribed, better adherence to the treatment plan, and more awareness of possible side reactions.

Every doctor has had patients who fail to finish antibiotic regimens because they feel better. Some patients assume that if one tranquilizer or analgesic is good, two may be twice as good. Still others fail to report dizziness while on antihypertensive therapy —and so on.

Problems like these might arise less often if the patient received written information in addition to verbal instructions. Some studies suggest that patients are more receptive to such materials, and they more often understand the verbal instructions and follow them, when inserts are used.

The Disadvantages

There are also some potential problems. Obviously, the inserts must be clearly phrased, without extraneous or complex detail. How much information is enough? How can it be kept current? Should all patients receive the same information? Should inserts be included with all drugs? Should only potential problems be listed or are patients better off with a "fair balance" presentation that describes usefulness as well as drawbacks?

These and similar questions require answers, since model inserts have yet to be properly developed and tested. Despite the need for these studies, the FDA is proceeding prematurely with inserts on selected products. We think the Congress is the only place where the matter can be given the proper legal status and direction, particularly since it represents a conceptual change in the legal, medical and social framework of the nation's prescription drug information system.

The Solution

The PMA believes that carefully-devised pilot studies of various kinds of inserts are needed. They should be developed and implemented with full participation by doctors, pharmacists, consumers, communications experts and the drug industry. Such studies will provide reliable pathways to follow, so that inserts will be useful aids to medical practice.

And particularly we think that you should be closely involved in this debate and in these studies and decisions. Otherwise, people with less experience and qualifications may control the purposes, content and use of a tool with considerable promise for improved patient care. It could make a difference in your practice tomorrow, and more importantly, in the health of your patients.

PMA
THE PHARMACEUTICAL MANUFACTURERS ASSOCIATION
1155 FIFTEENTH ST., N. W., WASHINGTON, D. C. 20005

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we're as close as your phone

Just use our Hot Line number 1-800-432-3587 for questions about:

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- Claims problems not resolved by regular correspondence
- Government Program policies
- Professional Relations matters that need a staff member's help.

but remember . . .

Calls on the "HOT LINE" cannot be transferred to a specific person because they do not come in on the regular Blue Shield switchboard. If you want to talk to a specific person, call through the regular number:

TOPEKA OFFICE - 1-913-232-1000
WICHITA OFFICE - 1-316-686-7263
DODGE CITY OFFICE - 1-316-225-0884

and ask for the person by name. However, you may call on the "HOT LINE" and leave a message for your assigned representative.

and don't forget . . .

When you need a visit to your office, you now have a specially assigned Professional Relations Representative who is ready to help. Call for your Blue Shield representative by name or leave your rep a message.

TOPEKA OFFICE - 1-913-232-1000
THE HOT LINE - 1-800-432-3587
WICHITA OFFICE - 1-316-686-7263
DODGE CITY OFFICE - 1-316-225-0884

Blue Shield
of Kansas

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TOPEKA, KANSAS 66601

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Practice Productivity Inc. is a national management consulting firm located in Atlanta whose only clients are physicians engaged in the private practice of medicine. Practice Productivity offers educational and motivational workshops in sound business concepts to physicians, medical office managers, and medical assistants. Practice Productivity also provides nation-wide individual consulting to physicians at their private practice.

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Registration Form

Name	Position	City Will Attend	Date Will Attend
1.			
2.			
3.			

Name of Practice: _____

Specialty: _____ Telephone: (_____) _____

Address: _____

City: _____ State: _____ Zip: _____

Full tuition fee of $ _____ is enclosed at $75 per registrant. Tuition includes course materials and luncheon and MUST ACCOMPANY THIS FORM. (There is a $10 handling fee deducted on all refunds for cancellations received at least one week in advance of course; no refund thereafter.)

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Councilor Reports

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The staff has mandated a required cardiac resuscitation program for every staff member.

The Douglas County Medical Society has organized and is sponsoring the Emergency Medical Technician program of the local ambulance service. We now have fully trained emergency medical technicians and cardiorespiratory technicians manning the ambulances.

The local medical auxiliary has plans underway to launch an annual flea market sale to obtain funds to donate to charitable projects, such as the South American Medical Relief Fund.

Overall, the main occupation of the physicians has appeared to be trying to keep up with the government, and everyone finds himself just a little bit behind.

ROBERT W. HUGHES, M.D., *Councilor*

DISTRICT 19

Council District 19, the Kansas Medical Society's newest district, is composed entirely of the members of the Southeast Kansas Medical Society. This organization is made up of approximately 50 physicians from five counties in Southeast Kansas. We meet at the Independence Country Club on the third Wednesday evening of each month. Each meeting consists of a social hour starting at 6:30, dinner, general business meeting, and usually a 30-40 minute scientific presentation by an invited guest or member. Members of the Auxiliary also conduct their meeting in a separate room during the general business meeting.

On February 15, 1978, the gavel was turned over by the retiring president, Dr. Charles Empson, Independence, to Dr. Donald White, Coffeyville. Other newly elected officers taking office were Dr. Bill Emmott, Independence, Vice President; Dr. Ed Robinson, Independence, Treasurer; Dr. Bob Haskins, Chanute, Secretary.

No serious problems have evolved during the preceding 12 months. It is with great relief that the District Councilor no longer has to serve as chairman of the Peer Review Committee.

ROBERT F. MOORE, M.D., *Councilor*

CLASSIFIED ADVERTISEMENTS

FOR SALE, office equipment used one year: 3M copier, model 215BG-VQCIII with service agreement; Lanier Action Line Transcriber and Receiver with foot pedal, one dictate station, and service agreement; Burdick EK5A EKG machine. Phone (913) 762-5050 or write P.O. Box 538, Junction City KS 66441.

COLLEGE HEALTH, Staff Physician, Emporia State University. Position available July 1, 1978. Call or write Wayne G. Parker, M.D., Director Health Services, ESU, Emporia, KS 66801; (316) 343-1200 ext. 224. An Equal Opportunity Employer M/F.

FOR SALE. Family practice in growing community of over 8000. Near hospitals and Wichita. Reply to Derby Clinic, RR#1, Box 41, Derby, KS 67037.

Mid-America Cancer Center Program seeking Associate Director for Cancer Outreach. Experience in clinical aspects of cancer, education, and community relations. Contact James T. Lowman, M.D., Director, Mid-America Cancer Center, KUMC, 39th and Rainbow, Kansas City, KS 66103.

PEDIATRICIAN needed for expanding multispecialty group of nine physicians, including one pediatrician. Excellent salary and fringe benefits. Opportunity to join Corporation after first year. City of 30,000; 35 miles from Kansas City and KUMC. Reply with resume to: David R. Barry, M.D., 500 Eisenhower Rd., Leavenworth, KS 66048.

CENTRAL KANSAS EMERGENCY STAFF POSITION, Great Bend, KS. Immediate opening. Evenings, Monday-Thursday. Moderate to low patient visits. $52,000/yr, paid malpractice, relocation allowance, liberal benefits. Send CV to John Crewes, M.D., Chase Stone Center, Holly Sugar Bldg., Suite 440, Colorado Springs, CO 80903, or call toll-free 1-800-325-3982, ext. 220.

HEALTH RESOURCES has immediate practice opportunities for general practice and family physicians, two physicians interested in geriatric medicine, and an ENT. Other primary care and surgical openings are available July 1. Please send CV in confidence to: Dr. Ronald Hammerle, Health Resources, Ltd., Box 12621, Kansas City, MO 64116.

ASPEN MUSHROOM CONFERENCE. Identification of edible, poisonous & hallucinogenic mushrooms; treatment of mushroom poisoning. Microscopy. Novice & advanced courses. AMA category 1. August 13-18, 1978. Wildwood Inn, Snowmass-at-Aspen, Colorado. Contact Beth Israel Hospital, 1601 Lowell Blvd., Denver, CO 80204. (303) 825-2190, ext. 359.

INTERNIST & FAMILY PHYSICIAN—Established need for both an internist & family physician to join 7-man group in beautiful NW Iowa. New clinic bldg. of 10,000 sq. ft. is located next door to 88-bed county hospital. Unusually progressive community of 10,000 offers 3,000-acre lake, 85 acres of parks & recreation, local liberal arts college, and many family interest features. Generous salary with incentive, malpractice insurance, liberal vacation & seminar time, partnership in 1 year. Contact D. A. Pritchard, Administrator, Buena Vista Clinic, 620 Northwestern Drive, Storm Lake, IA 50588.

FOR SALE: Examining table, two tonsillectomy sets, obstetric stethoscope, minor surgical set, base of postage meter. Contact: E. P. Wereres, M.D., 662 New Brotherhood Bldg., Kansas City, KS 66101.



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next breath...

he's active
he's effectively
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high theophylline for effective around-the-clock therapy

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Today's more efficient usage of theophylline includes individualizing dosage and monitoring serum theophylline levels. The usual recommended dosages of Quibron are: Adults—1 to 2 capsules or tablespoonfuls every 6 to 8 hours; dosage may be cautiously adjusted upward when necessary to a maximum of 2000 mg theophylline per 24 hours. Children under 12—4 to 6 mg theophylline per kg/body weight every 6 to 8 hours; dosage may be cautiously adjusted up to 9 or 10 mg/kg every 6 hours.

Now, for the asthmatic who requires high-dose theophylline therapy for therapeutic serum concentrations

Mead Johnson Pharmaceutical Division announces

QUIBRON®-300

Each capsule contains 300 mg theophylline (anhydrous) and 180 mg glyceryl guaiacolate (guaifenesin)

For Brief Summary, please see the last page of this advertisement.

QUIBRON®-300

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The new high-dose theophylline capsule... for dependable theophylline therapy when products of lower dosage do not adequately control asthma symptoms.

Specially formulated

...for optimal efficacy

Quibron-300 is appropriate therapy for asthma patients whose symptoms are not adequately controlled on lower doses of theophylline, particularly for patients whose theophylline dosage has been adjusted upward to achieve therapeutic serum levels. In one study,[1] an average peak increase in FEV_1 of 35% was demonstrated after a single dose equivalent to one Quibron-300 capsule, and significant improvement in this pulmonary function lasted for nearly eight hours after administration.

...for optimal predictability

One Quibron-300 capsule q6-8h yields therapeutic serum levels (10-20 mcg/ml) in many adults. With a single dose, more than 75% of patients achieved serum levels potentially providing clinical benefit (5-15 mcg/ml). Half-life of theophylline varies widely from patient to patient, making monitoring of theophylline therapy important. Patient response may be monitored clinically if blood levels are not available as long as dosage does not exceed 1200 mg in 24 hours for adults.

...for optimal dosage convenience

The simple, convenient dosage of new Quibron-300—one capsule every six to eight hours—makes it easy for patients to comply with high-dose regimens often required to achieve therapeutic serum levels. Quibron-300 capsules may provide maximum therapeutic value with maximum convenience. In fact, the switch from a low-dose to a high-dose regimen may be accomplished by merely switching capsules, by stepping up to Quibron-300 capsules.

...for minimal theophylline side effects

Adverse reactions to theophylline are related to serum levels and are usually not a problem at concentrations below 20 mcg/ml. Of 45 patients studied[1] after a single dose, only seven reported adverse reactions. The most common reaction was a feeling of lightheadedness by three of these seven patients.

Reference 1. Data on file. Mead Johnson Pharmaceutical Division.

Indications: For the symptomatic treatment of bronchospastic conditions such as bronchial asthma, asthmatic bronchitis, chronic bronchitis, and pulmonary emphysema.

Dosage: **Quibron**—Adults: 1-2 capsules or 1-2 tablespoonfuls elixir every 6-8 hours. Children under 12: 4-6 mg theophylline/kg body weight every 6-8 hours.

Quibron-300— Adults: 1 capsule every 6-8 hours.

Theophylline dosage may be cautiously increased to 2000 mg/24 hour in adults and 9 or 10 mg/kg every 6 hours in children. Monitoring of serum theophylline levels at higher dosages is recommended.

Precautions: Do not administer more frequently than every 6 hours, or within 12 hours after rectal dose of any preparation containing theophylline or aminophylline. Do not give other xanthine derivatives concurrently. Use in case of pregnancy only when clearly needed.

Adverse Reactions: Theophylline may exert some stimulating effect on the central nervous system. Its administration may cause local irritation of the gastric mucosa, with possible gastric discomfort, nausea, and vomiting. The frequency of adverse reactions is related to the serum theophylline level and is not usually a problem at serum theophylline levels below 20 μg/ml.

How Supplied: Quibron Elixir: Bottles of 1 pint and 1 gallon. Quibron Capsules: Bottles of 100 and 1000 and unit-dose packs of 100. Quibron-300 Capsules: Bottles of 100.

Mead Johnson PHARMACEUTICAL DIVISION

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MJL 7-4280

ROCHE

For recurrent attacks of urinary tract infection in women

Bactrim™ DS Double Strength Tablets



Each tablet contains 160 mg trimethoprim and 800 mg sulfamethoxazole.

Just one tablet b.i.d. for 10 to 14 days

- Action at urinary/vaginal/lower bowel sites helps eliminate reservoirs of infecting organisms
- Distinctive antibacterial action plus wide spectrum helps eradicate recurrent UTI
- Low incidence of bacterial resistance in community practice
- Convenient *b.i.d.* dosage provides day-and-night antibacterial control
- Contraindicated during pregnancy and the nursing period. During therapy, maintain adequate fluid intake; perform CBC's and urinalyses with microscopic examination.

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* **It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination.** *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

The recommended quantitative disc susceptibility method (*Federal Register,* 37:20527-20529, 1972) may be used to estimate bacterial susceptibility to Bactrim. A laboratory report of "Susceptible to trimethoprim-sulfamethoxazole" indicates an infection likely to respond to Bactrim therapy. If infection is confined to the urine, "Intermediate susceptibility" also indicates a likely response. "Resistant" indicates that response is unlikely.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older:

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension,* containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

ROCHE

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

Please see back cover.

Serials Librarian SUB
U. of California Medical Center
San Francisco, Calif. 94143

Her next attack of cystitis may require the Bactrim™ 3-system counteratta



ROCHE

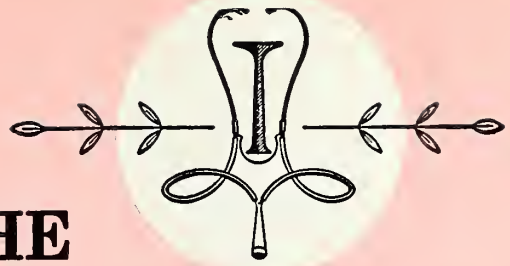
Bactrim has shown high clinical effectiveness in recurrent cystitis as a result of its wide spectrum and distinctive antimicrobial action in the urinary, vaginal and lower intestinal tracts.

The probability of recurrent urinary tract infection appears to be enhanced by the establishment of large numbers of *E. coli* or other urinary pathogens on the vaginal introitus. The trimethoprim component of Bactrim diffuses into vaginal fluid in effective concentrations, thus combating migration of pathogens into the urethra.

Studies have shown that Bactrim acts against *Enterobacteriaceae* in the bowel without the emergence of resistant organisms. Thus, Bactrim reduces the risk of introital colonization by fecal uropathogens. It has *no* significant effect on other normal, necessary intestinal flora.

Bactrim fights uropathogens in the urinary tract/vaginal tract/lower intestinal tract

Please see reverse side for summary of product information.



A.C.S. Issue

THE Journal OF THE Kansas Medical Society

Journal

MAY
1978

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The JOURNAL of the KANSAS MEDICAL SOCIETY

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

Librax®

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium Br.

Please consult complete prescribing information, a summary of which follows:

Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma, prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

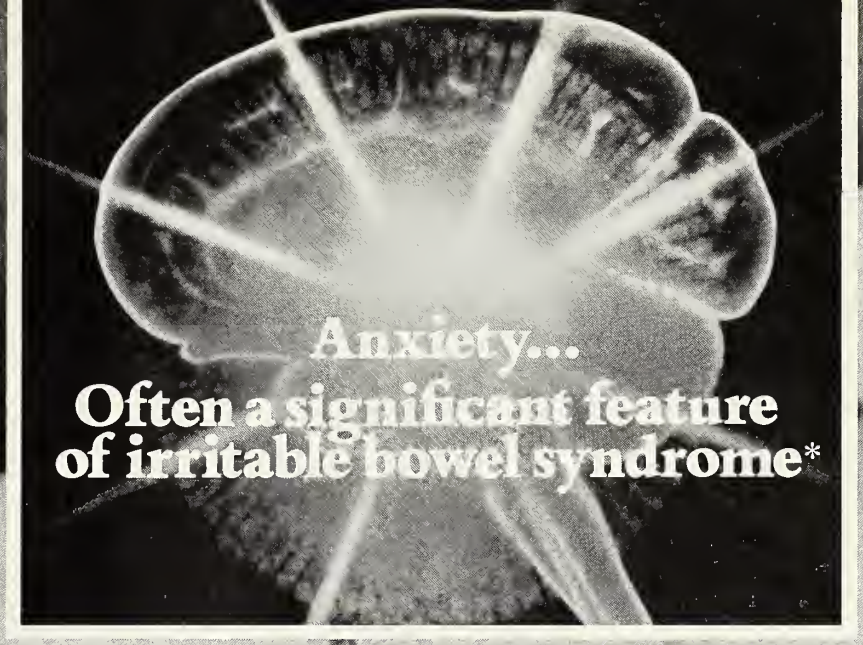
As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated, avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, i.e., dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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Please see brief summary of prescribing information on following page.

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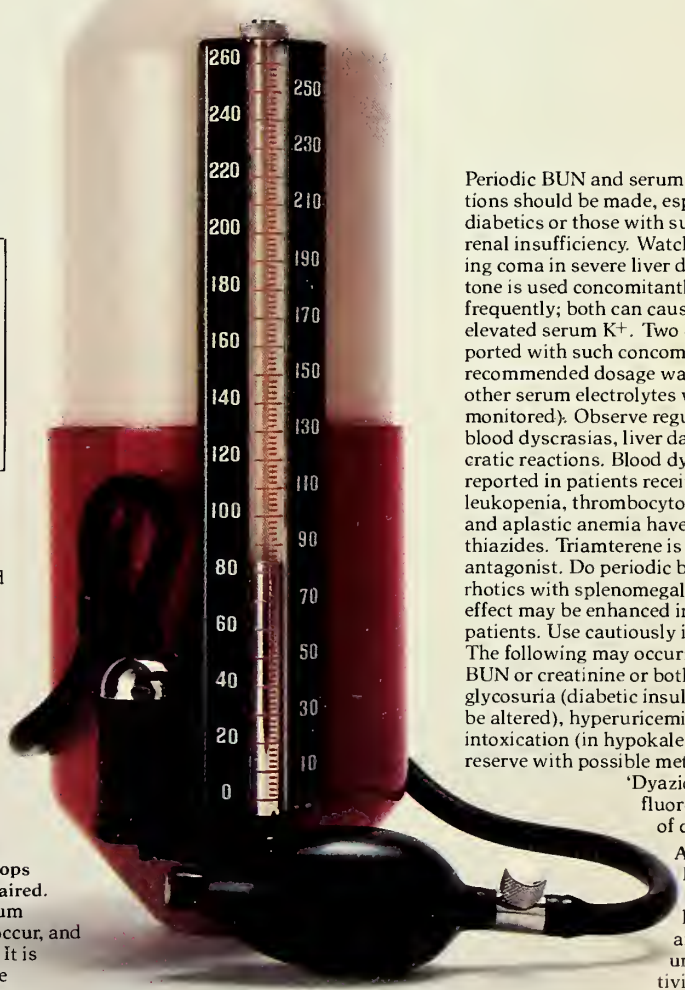
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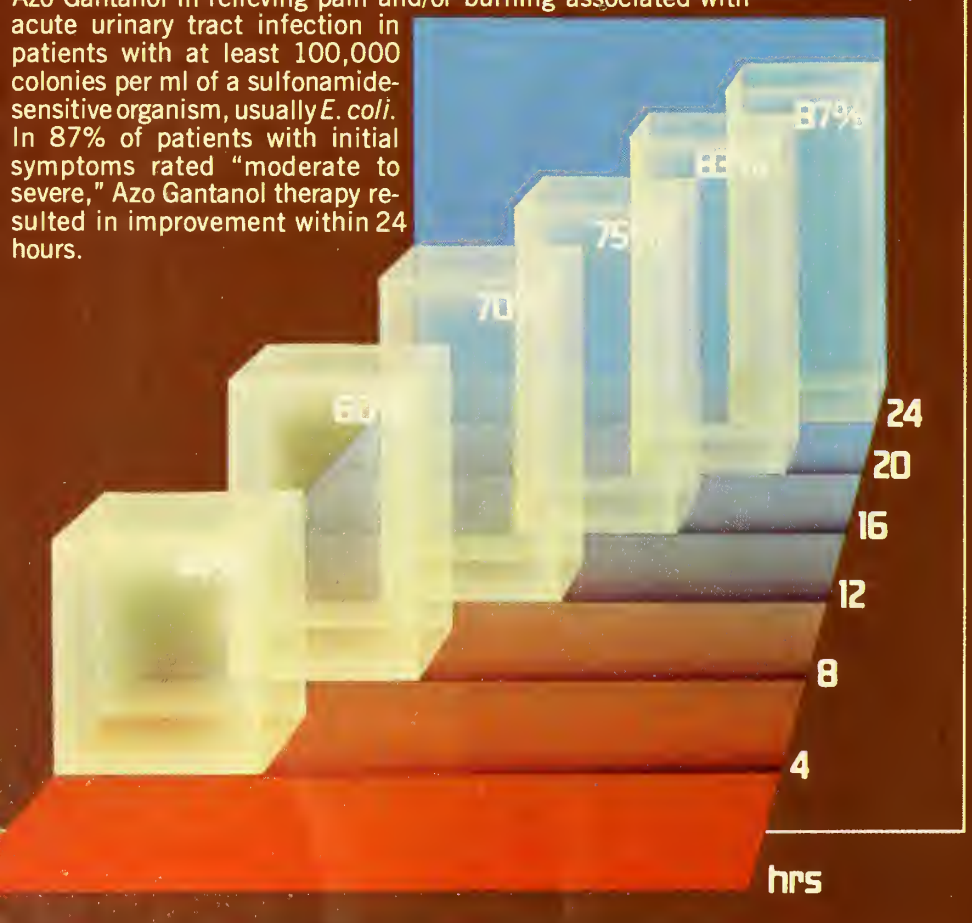
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Contraindications: Children below age 12; sulfonamide hypersensitivity; pregnancy at term and during nursing period; because Azo Gantanol contains phenazopyridine hydrochloride it is contraindicated in glomerulonephritis, severe hepatitis, uremia, and pyelonephritis of pregnancy with G.I. disturbances.

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Precautions: Use cautiously in patients with impaired renal or hepatic function, severe allergy, bronchial asthma; in glucose-6-phosphate dehydrogenase-deficient individuals in whom dose-related hemolysis may occur. Maintain adequate fluid intake to prevent crystalluria and stone formation.

Adverse Reactions: *Blood dyscrasias* (agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia); *allergic reactions* (erythema multiforme, skin eruptions, Stevens-Johnson syndrome, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis); *G.I. reactions* (nausea, emesis, abdominal pains, hepatitis, diarrhea, anorexia, pancreatitis and stomatitis); *CNS reactions* (headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo and insomnia); *miscellaneous reactions* (drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon). Due to certain chemical similarities with some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia. Cross-sensitivity with these agents may exist.

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TRACE ELEMENTS

Essential trace elements, like vitamins, are required by the animal organism in very small amounts (in the range from micrograms to milligrams). In addition, trace elements, like vitamins, act as catalysts of biochemical reactions. However, although these two categories of micronutrients are very different from each other in their chemistry (simple inorganic vs. complex organic molecules), they are quite similar in a nutritional sense. As a result, nutritional problems that affect vitamins are likely to affect the trace elements. Also, they have in common the public health problem of *marginal* deficiencies. Since life-threatening diseases, such as pellagra and severe iron deficiency anemias have been virtually eliminated through nutrition intervention, the major remaining problem relates to the definition of the "optimal" human requirement . . . this definition is the major challenge of trace element research.

Mode of Action

Trace elements act as catalytic or structural centers of enzymes (iron, zinc, copper, molybdenum) or of a vitamin (cobalt in vitamin B_{12}) or as a constituent or activator of a hormone (iodine in thyroid hormones; chromium with insulin). This explains why very low concentrations of trace elements have profound effects in the organism. They are small catalytic centers of much greater molecules, which act as catalysts by themselves. For example, one atom of chromium is believed to initiate the action of insulin. This corresponds to a mass ratio of 52:6000. The concentration of insulin in the blood in the fasting state (approximately 10 micro-units/ml) corresponds to 400 picograms or 0.0000000004 g/ml. Thus, the equivalent concentration of chromium would be 1/100 of this. This calculation emphasizes the great demands that modern trace element research puts on analytical methodology.

Availability

The most reliable analytical result does not give all the information that the nutritionist requires in order to assess the quality of diets. It is of the utmost importance to realize that the adequacy of trace element nutrition is not only a matter of quantity but also of quality. Depending on the chemical form in which an element is present in the diet and depending on the presence of a large number of poorly defined dietary factors, the availability of a trace element can range from 0 to 100%. For example, zinc deficiency occurs in people in Iran whose zinc intake is greatly in excess of the Recommended Dietary Allowance in the U.S.A. In addition, depending on the form in which iron is present in the diet and on the presence of meat or vitamin C, the biological availability of iron can vary by a factor of 10.

Cobalt in inorganic form is nutritionally useless to monogastric species, including man. Similarly, chromium must be present in the form of an organic complex with nicotinic acid, termed glucose tolerance factor to be assimilated. Similar dependencies on the chemical form may well exist for copper, selenium and silicon.

In addition to the dependence on chemical form, the biological action of trace elements is also determined by interactions with other minerals, trace elements and macronutrients in the diet. For example, calcium is known to decrease the availability of many trace elements and, therefore, to increase their dietary requirements. In animals and probably in man, the copper requirement depends strongly on the presence of molybdenum and sulfur. A given copper intake from forage can be optimal, deficient or result in copper toxicity, depending on the amount of molybdenum present.

Of great potentail significance is the interaction of selenium with heavy metals, for example, cadmium and mercury. Selenium protects against the acute and chronic toxic effects of these metals; thus, a given concentration of cadmium or mercury in the environment can be toxic or harmless, depending on the concentration of selenium present. It can be assumed that the selenium requirement, in turn, depends on the concentration of these heavy metals in diet and environment. There are other metal interactions too numerous to be discussed in this article.

An outstanding example of metal interaction with macronutrients in the diet is that of iron and meat. The presence of meat in an iron-containing meal significantly increases the absorbability of nonheme iron. This effect is not due to the protein or amino acid content; the nature of the absorption enhancing "meat factor" is still unknown. Dietary fiber decreases the availability and increases the requirement for calcium, magnesium and, possibly, zinc. Dietary protein increases the requirement for calcium. Phosphates, phytates, antacids, coffee and tea are known to decrease significantly the availability or absorption of several trace elements. These examples strongly suggest that it is impossible to establish a quantitative "requirement" for trace elements unless the exact composition of a particular diet is known. In general, it can be stated that animal products are good sources of available iron, zinc and chromium and that these metals are much better utilized from these sources that they are from vegetables in which they may occur in high concentrations. The Recommended Dietary Allowances of the Food and Nutrition Board are predicated on the consumption of a varied diet. Any deviation from the concept of variety, for example, a vegetarian diet, would increase the dietary requirement in order to compensate for the

decreased availability of trace elements from vegetable sources.

These considerations are important guidelines for food enrichment and fortification. The nature of the fortified food is as important as the amount and type of the fortification element.

Categories

For convenience of discussion, the trace elements can be divided into three categories: the first comprises those with a well-defined human requirement, such as iron, zinc and iodine. The second consists of those for which a human requirement is known to exist but has not yet been quantified. To this category belong chromium, manganese, copper, selenium and molybdenum. The "new" trace elements form the third category. Fluorine, silicon, vanadium, nickel, arsenic, tin and possibly cadmium have all been shown to be required by animal species. However, quantitative requirements are not known and the difficulties of analysis prevent even an approximate assessment of requirement and nutritional status in man.

1. Iron, zinc and iodine. Recommended Dietary Allowances have been established for these three minerals; 10 and 18 mg of iron/day are recommended for adult men and women of childbearing age, respectively. The actual dietary intake is a mirror image of the R.D.A.'s; men consume an amount close to the allowance for women and women's intakes are close to the allowance for men. The average iron content of representative diets in the U.S.A. is approximately 6 mg/1000 kcal. The actual iron intake depends on the energy consumption, which is limited in the modern woman. Although the iron enrichment program in the past has not resulted in a complete reduction of iron deficiency, the new concept of dietary influences on the nonheme iron pool promises to become a powerful tool for the improvement of the iron nutritional status of women in the U.S.A.

The R.D.A. for zinc is 15 mg/day for adults. As previously discussed, the actual zinc requirement depends on the composition of the diet. Since the availability of zinc in animal foods is higher than in vegetable foods, the R.D.A. of 15 mg is predicated on the consumption of a varied diet. The actual intake from typical U. S. diets falls short of this figure and is in the range of 10-15 mg (R.D.A.-N.A.S.) depending on energy and protein intake. This discrepancy between actual and recommended intakes is of concern to nutritionists because zinc is rapidly utilized and is of great importance in essential biochemical and physiological functions.

The R.D.A. for iodine is 100-140 μg/day for adults. This requirement is readily obtained from the typical U. S. diet and intakes much in excess of this figure are not uncommon. The iodization of table salt has resulted in the elimination of practically all iodine-deficiency goiter in this country; the incidence of "residual goiter" is unrelated to deficient iodine intake. Substantial amounts of iodine in the environment come from adventitious sources, such as dough oxidizers, disinfectants used in the dairy industry, alginates and coloring agents. This situation is of concern to the nutritionist because recent increases of iodine intakes have resulted in an increased incidence of toxic goiter in some countries.

2. Chromium, manganese, copper, selenium and molybdenum. Deficiencies of chromium and copper have been described in human subjects abroad suffering from protein-energy malnutrition and in the U.S.A. in patients on total parenteral nutrition. Marginal deficiency of both elements may occur in the U. S. population. Chromium-responsive cases of impaired glucose tolerance have been described in U. S. subjects. The dietary chromium requirement has not yet been quantified; it may be between 50 and 200 μg/day. The dietary requirement for copper, on the other hand, may be close to 2 mg/day on the basis of several balance studies. The actual intake as determined in recent surveys is considerably less than 2 mg. Self-chosen diets of young persons contained an average of only 1 mg/day. Although the typical signs of pronounced copper deficiency do not exist in the U. S. population, the uncertainty of a sufficient copper intake must be of concern.

The human requirements for mangenese, selenium and molybdenum have been estimated on the basis of balance studies. They may be at 2.5 mg and 75 μg and 150μg, respectively. Molybdenum deficiency is not known in human subjects. One case of possible manganese deficiency under experimental conditions has been described and the possibility of selenium deficiency is under investigation in New Zealand, where dietary intakes are extremely low. These considerations point out potential problems of marginal deficiencies in some population groups but much more research is necessary before the nutritional status of the U. S. population can be adequately assessed.

3. The "new" trace elements. Deficiencies of these elements have been induced in experimental animal species by feeding highly refined, purified diets, purified water and by keeping the animals in an environment that allowed exclusion of metal pollution. Signs of deficiency range from gross bone deformities (silicon) to impaired growth and reproduction to unexplained sudden death (arsenic). Although the practical implications of these findings for human nutrition are uncertain at the present time, some theoretical statements can be made. If a trace element is found to have an essential role in one or more mammalian species, the probability is substantial for an

(Continued on page 230)

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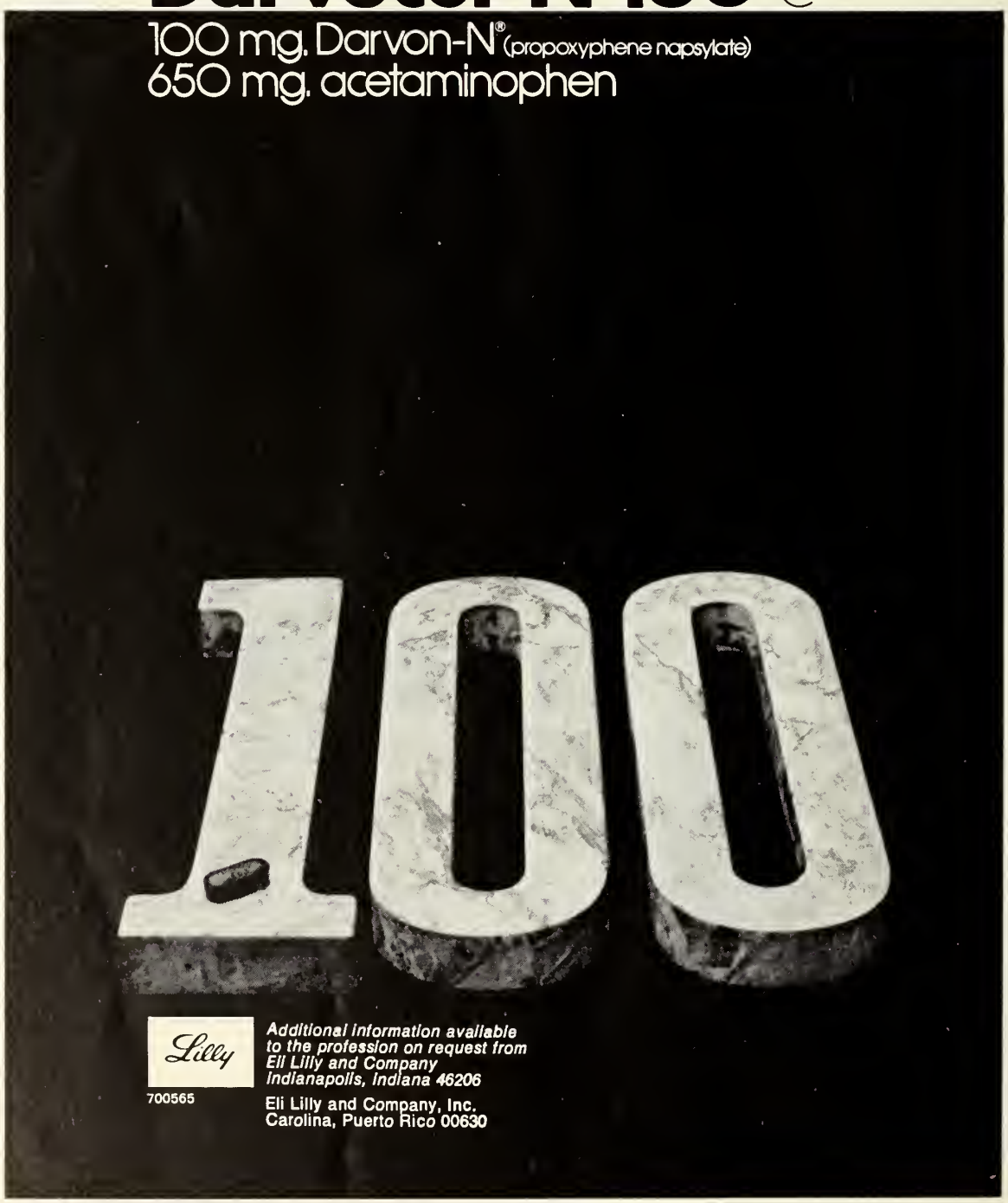
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Urinary Tract Injury

Management of Renal and Bladder Trauma

JOHN D. MOYER, M.D., ***Topeka***

Methods of urographic assessment in cases of known or suspected injury to the urinary system are evaluated and discussed. A rationale is presented for utilization of procedures which have resulted in improved renal salvage and elimination of unnecessary surgery.

SERIOUS INJURIES to the urinary tract are sufficiently common that large series have been collected and studied over relatively short periods of time in many centers with active trauma units. The consensus has evolved that many such injuries are best managed by rapid, accurate radiologic assessment and non-operative management. Since most urinary tract injuries are associated with concurrent and often more life-threatening injury to other organ systems, appropriate priority must be established; but in virtually all situations, crucial urographic studies can be obtained with very little time and effort. These studies are very often pivotal in early and long-term patient management.

Kidney Injuries

The past quarter century has seen a shift from surgical exploration for virtually all cases of known or suspected kidney injury to almost the opposite extreme. The routine use of intravenous pyelography (IVP), for both early evaluation and follow-up, has demonstrated conclusively that most (75%,[1] 86%[2]) established renal injuries from blunt trauma managed without surgery will heal without residual deformity. On the other hand, penetrating injuries should usually be explored because: (1) the early IVP can be negative in the face of major injury; (2) the incidence of associated injury to other organs is very high; and (3) late complications are significant. Maximal renal salvage can be achieved with proper surgical technique.

Radiographic Assessment

Ideally, all patients with severe trauma to the chest, flank, or abdomen should receive early IVP with tomography if possible. The majority of these will have gross or microscopic hematuria, but this cardinal sign may not be present in very severe insults, such as avulsion of the upper collecting system or renal pedicle and traumatic thrombosis of the renal artery. This study is best obtained in the radiology department, but very useful information can be obtained by portable x-ray units in the emergency room or operating room. Severe hypotension (systolic blood pressure below 70 mg Hg) and known severe reaction to the contrast medium are perhaps the only contraindications. On occasion, the IVP should be delayed until urographic assessment of the lower urinary tract has been completed (see below). Arteriography may be the initial procedure of choice, especially when liver or spleen injury is suspected. An

Presented at the Annual Meeting of Kansas Chapter, American College of Surgeons, Topeka, November 12, 1977.

infusion of diatrizoate or iothalamate (*e.g.,* Renografin 60%) 1 cc/lb, 200 cc maximum over 5-10 min. is the method of choice. A delay in this procedure may yield misleading information because of parenchymal compression from intracapsular hematoma or poor renal visualization due to overlying gas from meteorism, or retroperitoneal bleeding from a non-renal source.

The IVP should be followed, or in some situations replaced, by arteriography. The elegant studies of Lang have shown by renal arteriography how misleading a "normal" IVP can be. Especially noteworthy are the high incidence of arteriovenous fistulae and parenchymal separation undetected by IVPs. The viability of the margins in a parenchymal separation may be of value in determining treatment. Intraoperative renal arteriography has been investigated experimentally.

The radioisotopic renal scan is of questionable value in the acute situation, but may prove useful as a follow-up study. Finally, the time honored retrograde pyelogram is rarely indicated; probably this procedure should be utilized only just prior to exploratory surgery in cases of suspected disruption of the collecting system.

Management

The number and type of classification of kidney injuries must surely approximate the number of papers and chapters on this subject.[1-6] Treatment should be dictated by a combination of experience, clinical judgment, the expertise of the surgeon(s) involved and the quality of radiological assessment, not by "type" or "classification." All renal injuries are worrisome, so good working diagnostic categories might be worrisome, very worrisome, or extremely worrisome. In more clinical terms, *mild, moderate,* and *critical* renal injuries will be discussed, keeping in mind that there is considerable interchange among these areas.

Patients with mild kidney injury will have a history of flank trauma, some degree of hematuria, and a normal or near normal IVP. Approximately one in five of these will have a parenchymal tear instead of a contusion. Appropriate treatment consists of absolute bedrest for seven-ten days, or longer if hematuria persists. Since most of these patients are young, sedation may be necessary. Daily urinalyses are mandatory, with vital signs, flank examination, and other monitoring as indicated. Activity must be rather severely restricted for about a month post injury to minimize the incidence of delayed or secondary bleeding.

The management of patients with very worrisome or moderate kidney injury have vigorous advocates in both surgical (aggressive)[4] and non-surgical (conservative)[2] camps. This group varies from 10-40 per cent of all renal injuries and, in general, shows evidence of parenchymal laceration by exhibiting a flank mass, or tenderness, with evidence of significant blood loss. The IVP clearly reveals impairment, with extravasation of contrast medium, often extracapsular. In the majority of these patients, associated injuries will be a key factor in determining the need for surgical exploration. Factors relating to the kidney injury include persistent bleeding after about 24 hours, clinical instability after transfusion of three-four units of blood, known solitary kidney or coexisting renal disease, expanding or pulsatile flank mass, and deterioration of the IVP after 24-48 hrs of observation. The paramount principles involved in successful renal salvage if surgery is performed include optimal preoperative urographic assessment, a transperitoneal approach with early control of the vascular pedicle (see below), and careful, deliberate renal reconstruction.

The critical kidney injury is seen in a potpourri of trauma situations. These constitute about 5-10 per cent of all renal injuries, and are associated with a high mortality rate often attributable to concurrent head or chest injuries. Penetrating injuries dominate this area. The value of urography before or during surgical exploration cannot be overemphasized. The surgeon must be assured that the contralateral kidney is normal, or at least present and functioning. A shattered kidney will show impairment of visualization, total loss of renal architecture, or extensive extravasation. If arteriography demonstrates fragmentation along the lines of segmental blood supply, with the fragments in good proximity, and there is no expansion of the retroperitoneal hematoma during intra-abdominal surgery, conservative management has been shown to be a reasonable option. Under less favorable conditions expeditious nephrectomy may be indicated. Between these extremes, attempted renal repair depends on the status of the patient and surgical judgment. Scott and Carlton are preeminent in the field of emergent renorrhaphy. Their precepts of initial vascular control with bulldog clamps or vessel loops via the posterior parietal peritoneum before releasing the renal fossa tamponade, watertight closure of calyceal tears, debridement with accurate hemostasis, and coverage of the exposed parenchyma with renal capsule or free peritoneal grafts[7] have been acknowledged as instrumental in recently reported increased renal salvage rates.[8] The combination of nonvisualization of a kidney with little or no hematuria is usually diagnostic of an extremely critical injury, that of renal pedicle disruption or traumatic arterial thrombosis. Renal salvage in these situations depends largely on the expertise of the surgeon in vascular reconstruction. If arteriography shows flow past an arterial thrombosis, ancillary data from experience with renal artery embolism suggests that kidney recovery is quite likely without surgical intervention.

Urography may demonstrate preexisting renal disease predisposing to injury, notably carcinoma, hydronephrosis, pyelonephritis, renal ectopia, horseshoe kidney, or other anomalies. There are generally anecdotal cases managed according to circumstance and judgment, keeping in mind that delayed nephrectomy after the patient has stabilized and optimal diagnostic studies have been performed is an entirely valid mode of treatment. Antibiotic therapy is indicated in most such cases. Follow-up for kidney injuries managed without nephrectomy is essential to detect late complications, primarily delayed bleeding, hypertension, sepsis, and obstruction.

Injuries to the ureter are frequently associated with gunshot wounds.[9] Primary repair is usually possible. Debridement, spatulation of the viable ends and a tension free, watertight anastomosis are essential. If primary repair is untenable at the time of initial exploration, temporary diversion can be readily accomplished by catheter ureterostomy or nephrostomy. The many other considerations and approaches to ureteral or ureteropelvic junction injuries are beyond the scope of this paper.

Bladder Injuries

Until quite recently, emergent closure of bladder ruptures with suprapubic cystostomy was considered axiomatic in urologic surgery. A high mortality rate attendant to this injury was documented in war injuries, especially in the preantibiotic era. But here again emergent radiologic assessment and careful case selection have given credence to non-surgical management.

Radiographic Assessment

A properly executed cystogram will demonstrate bladder rupture and the type of extravasation whether intra- or extraperitoneal in virtually all situations. Water soluble sterile contrast medium is introduced by gravity instillations via an indwelling catheter. A film is taken after 100-150 cc is instilled and again after filling up to 400 cc. Of extreme importance is the post-drainage washout film to show extravasated material obscured by intravesical dye. In some situations, a preliminary retrograde urethrogram is indicated, easily obtained in the emergency situation using a Toomey or Asepto syringe or even a regular 50 cc syringe with an adaptor. These studies should generally be performed before intravenous pyelography.

Management

Most traumatic ruptures of the bladder occur with automobile accidents, especially in conjunction with heavy alcohol ingestion. About 10 per cent of pelvic fractures will have associated bladder injuries.[9] Very mild trauma will cause bladder ruptures, and even spontaneous ruptures are seen in alcoholics, patients with prostatic obstruction, detrussor sphincter dyssynergia, and other rare situations. If bladder injury is suspected, and the patient is unable to void, voids only a small amount of very bloody urine or has blood seeping from the urethra, a retrograde urethrogram is essential. The conventional approach of first passing a soft catheter is to be discouraged in such situations. First, even minor manipulation may turn a partial urethral tear into a complete disruption. Second, in rare but potentially catastrophic situations the catheter will simply drain urine leaking from a disrupted bladder neck into the pelvic space occupied by the catheter tip. If the urethrogram shows continuity and rectal examination indicates the prostate is in normal anatomical position, a 24 French Silastic Foley catheter may then be gently advanced and a cystogram immediately performed to establish the position of the catheter and to determine bladder rupture.

At this point, non-surgical management for bladder rupture may be elected using these criteria: (1) the catheter has passed easily; (2) the diagnosis of bladder rupture has been made within 12 hrs of injury; (3) there is no history of active urinary infection or complicating genitourinary disease; (4) the patient has a proven rupture and there is no other indication for emergent surgery; and (5) if the patient's condition deteriorates or there is reason to suspect that treatment is inadequate (*e.g.*, acidosis, rising blood urea nitrogen, sepsis), surgical exploration can be expeditiously performed.[10] Prophylactic broad spectrum antibiotics, appropriate supportive care, and at least 10 days of free catheter drainage are routinely employed in this nonoperative approach.

Patients who do not fulfill the above criteria must have emergent surgery. Evacuation of blood and urine, generous irrigation with an antibiotic solution (*e.g.*, cephalothin 2%), thorough exploration, especially of the bladder dome, watertight closure in layers, suprapubic cystostomy with a large bore catheter, and perivesical drainage are generally accepted surgical principles. The integrity of the distal ureter must be ensured with penetrating injuries. Uniformly good results can be expected.[9, 11, 12]

The treatment of urethral injuries is complex and controversial. There is an emergent situation, avulsion of the membranous urethra at the superior leaf of the urogenital diaphragm, which the general surgeon may be called upon to manage. The associated injuries and findings include severe pelvic fractures involving the pubic rami and ischium with wide separation of the pubic symphysis, inability of the patient to void even when the bladder is apparently distended, and a floating prostate detected by rectal examination. The urethro-

gram confirms the diagnosis. The simplest emergency treatment is suprapubic cystostomy with thorough deep pelvic drainage as advocated by Morehouse. The inevitable stricture is operated after an appropriate interval. Turner-Warwick[12] has utilized a more elegant approach that can be readily added to the surgeon's armamentarium. The immediate complication rate is reduced and subsequent urethral surgery is greatly simplified. The essentials of this approach include passing a large fenestrated urethral catheter across the defect into the bladder, traction applied by heavy sutures through the prostate apex, and special attention to adequate retropubic drainage, along with the usual suprapubic cystostomy. Do not engage in prolonged efforts with interlocking sounds or apply traction on the bladder neck with a balloon catheter.

Conclusions

Renal and bladder trauma can be evaluated in the acute situation by radiologic techniques yielding a high degree of diagnostic accuracy and requiring little or no loss of time. Accurate urographic assessment provides specific indications for conservative management of apparently severe injuries, and more importantly, allows the surgeon to anticipate the techniques he must employ for successful kidney salvage. Preservation of renal function with acceptable risk is the keystone in the management of genitourinary trauma. Safe, reliable temporizing procedures have been developed to provide urine drainage until the trauma victim can undergo reconstructive surgery for collecting system injuries.

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The APUD Concept

Ectopic Endocrine Syndromes

STANLEY R. FRIESEN, M.D., Ph.D.,* *Kansas City, Kansas*

Ulcerogenic syndrome, in which the pancreatic tumor secretes a polypetide hormone, gastrin, which in turn provokes massive hypersecretion of acid resulting in fulminating and recurrent ulcer disease, was described in 1955 by Zollinger and Ellison.[1] The fact that gastrin, a native hormone of the gastric antrum, could emanate from the pancreas was a totally unexpected finding and a clear demonstration of an ectopic endocrinopathy.

Since that time, more and more endocrine cells and syndromes have been found and described, so that now the largest part of the neuroendocrine system resides in the gastro-entero-pancreatic (GEP) system.[2] Furthermore, many of the same hormones of this system have also been found to be secreted from the hypothalamus.

The cells that secrete these polypeptide and amine hormones have now been designated as APUD cells, an acronym denoting their common cytochemical characteristics: Amine Precursor Uptake and Decarboxylation.[3] These cells all take up precursor amines, and store and secrete amines and polypeptide hormones. They have these common characteristics because of their apparent common embryologic background; they arise from the neuro-ectoderm of the embyro and migrate to their location in the entoderm of the gut, pancreas, and other endocrine sites where they function as expected in their new habitat in an entopic normal fashion.

However, two general abnormal mechanisms may lead to ectopic function in which hormones may be liberated from unexpected sites: (1) if migration of APUD cells is arrested developmentally, these ectopically located cells may function in mesodermal areas; or (2) if neoplasia develops, these cells may revert to a very primitive precursor state by a process of derepression and secrete any of the APUD cell hormones, irrespective of their location.[4] Thus, a malignancy of the pancreatic islets, by reverting to a totipotential precursor state, may secrete any of the polypeptide hormones, such as adrenocorticotrophin (ACTH), gastrin, or even a polypeptide having the activity of parathyroid hormone, as is being reported elsewhere in this issue of the *Journal* by Drs. Bosiljevac and Robertson. The molecular size of this latter ectopic hormone may be larger than that of the normal parathyrin, and thus be more difficult to assay than the normal entopic hormone.

The migratory dynamics of the APUD cells are explored and the endocrinopathic effects are described.

The scientist who has been most instrumental in developing the APUD concept is Professor A. G. E. Pearse, London, a histochemist and cytologist. His early descriptions of these endocrine cells have been confirmed. The APUD endocrine cells have a common appearance of being round or oval with large nuclei and with secretory granules in the cytoplasm, which become evident by light, electron, and immunofluorescent microscopy. These cells normally secrete a single amine or polypeptide hormone to maintain normal neurohumoral homeostasis. However, when they become hyperplastic they may secrete abnormally high levels of their hormones, as is seen in the multiple endocrine adenopathy (MEA) syndromes; or they may become adenomatous, such as is seen in insulinomas of the pancreas; or they may become malignant and metastasize, forming multiple foci of hypersecretion, in which case they may secrete multiple hormones and even demonstrate ectopic function.

If ectopia becomes apparent in patients, one may assume that the tumor is malignant and is located at any site other than where it is normally expected to be. In ectopia, the radioimmunoassay of the polypeptide which emanates from its normal site will most likely give a normal or low (suppressed) value. Thus, in ectopic hyperparathyrodism from an endocrine carcinoma of the

(Continued on page 210)

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Endocrine Surgery

Pancreatic Islet Cell Tumor Presenting as Hyperparathyroidism

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HYPERCALCEMIA has frequently been reported with malignancy. One postulated cause has been the production of a parathyroid hormone-like substance from the tumor tissue. Reports of hypercalcemia and secretion of an ectopic parathyroid hormone-like substance in association with an islet cell tumor of the pancreas are rare.[1-3] This paper presents such a case of hypercalcemia presumably due to a parathyroid hormone-like substance associated with an islet cell tumor, and reviews the literature on the subject.

Case Report

A 33-year-old white female became aware of a left-upper quadrant mass, 20-lb weight loss, amenorrhea, polyuria, polydipsia, and nocturia four months prior to admission. These symptoms prompted her to consult her primary physician. Pertinent physical findings revealed normal vital signs and an epigastric mass which was nontender, firm, and fixed in the left-upper quadrant. This extended from the midline of the epigastrium to the lateral abdominal wall at the left costal margin. Initial laboratory data included a normal hemogram and SMAC with the exception of an elevated alkaline phosphatase with an increase in liver and bone bands. Initial laboratory findings revealed the following results: serum calcium, 14.2 mg/100 ml, and serial serum calcium studies confirmed hypercalcemia in this range; phosphorus, 1.5 mg/100 ml, repeated studies confirmed hypophosphatemia; fasting blood sugar, 136 mg/100 ml; two-hr postprandial blood sugar, 113 mg/100 ml; urine calcium, 1,129 mg/24 hr (100-300 normal range); parathyroid hormone assay, less than 390 pg/ml (normal, 2,000 pg/ml); CEA, urine VMA, urine catecholamines, serum amylase and lipase, and two-hr urine amylase and lipase were normal; serum cortisol, T-4, TSH, total estrogen, and urine 17 Keto- and 17 Hydroxysteroids were normal; chest x-ray, EKG, bone scan, and liver scan results were normal; abdominal sonogram revealed a large mid-abdominal mass separate from the kidney and the spleen; GI studies confirmed the mass in a retrogastric position and arteriography demonstrated tremendous vascularity of the lesion.

Following preoperative evaluation, bowel prep was completed and surgical exploration performed. A very large neo-vascular tumor of the tail of the pancreas was identified contiguously involving the posterior wall of the stomach, transverse colon and mesocolon, and the spleen. Marked venous dilatation and neo-venous vascularization was evident in the draining gastroepiploic system. An en bloc subtotal pancreatectomy, subtotal gastrectomy, splenectomy, and segmental transverse colectomy was performed with appropriate gastroenterostomy and colocolostomy. No clinical evidence of metastatic intraabdominal disease was evident except for the primary tumor and local organ involvement. Histologic evaluation revealed a pancreatic islet cell tumor.

A rare case of hypercalcemia and secretion of a parathyroid hormone-like substance in association with an islet cell tumor is reported and a review of the literature is presented.

The postoperative course was complicated by atelectasis, pneumonia, thrombophlebitis, and sepsis; however, the patient recovered during a one-month hospitalization. Postoperatively the serum calcium dropped to 11 mg/100 ml and phosphorus levels returned to normal (3.4 mg/100 ml). Immediate postoperative parathyroid hormone studies remained less than 390 pg/ml. No insulin was required during the postoperative period. At three month follow-up the patient had gained 18 lbs and normal menstruation had resumed. Fasting blood sugar was 90 mg/100ml; serum calcium, 9.6 mg/100 ml; serum phosphorus, 3.4 mg/100 ml; urinary calcium 17 mg/24 hr. Parathyroid hormone assay was 2,040 pg/ml (within normal limits) at this time. Follow-up at six months and again at one year demonstrated no clinical evidence of recurrent or metastatic disease.

Discussion

In 1941, Albright[4] suggested that some tumors of

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non-parathyroid origin might produce substances with biologic activity similar to parathyroid hormone. Sherwood[5] believed that certain signs suggest the production of such a hormone, such as an absence of skeletal metastases, a low serum phosphorus, and normal parathyroid glands at surgery or autopsy. After removal of the tumor, an increase in serum calcium occurs with recurrence of the tumor. Despite these signs, it is often difficult to differentiate hypercalcemia of malignancy with ectopic parathyroid hormone and primary hyperparathyroidism.

Pseudohyperparathyroidism has been defined by Lafferty[6] as a syndrome of hypercalcemia and hypophosphatemia occurring in patients who have nonparathyroid neoplasms without osseous metastases. From the literature, he gathered 50 cases meeting at least one of his criteria: (1) absence of roentgenographic and postmortem evidence of bony metastasis, and failure to find parathyroid adenomas at surgical exploration or careful postmortem examination; (2) significant reduction of serum calcium after resection of the neoplasm; or (3) positive parathyroid hormone immunoassay of the neoplasm extract. Lafferty found that pseudohyperparathyroidism was most frequently seen with hypernephroma and bronchogenic carcinoma, which constituted about 60 per cent of the cases he reported. He considered the synthesis of a parathyroid hormone-like polypeptide to be the most likely mechanism for the syndrome. Other causes of hypercalcemia—such as vitamin D intoxication, milk alkali syndrome, metabolic bone disease, multiple myeloma and leukemia, sarcoidosis, hyperthyroidism, myxedema, adrenal insufficiency, and hypercalcemia of immobilization—must be ruled out by history and physical examination and appropriate laboratory studies. If this is done, then the syndrome of pseudohyperparathyroidism vs. primary hyperparathyroidism must be differentiated.

Lafferty suggested that certain aspects of the patient's history and certain laboratory tests might be helpful in this differentiation. He thought the hypercalcemia was more rapid at onset of malignancy than with primary parathyroid disease, and that renal disease and osteitis fibrosa cystica was more common in patients with primary parathyroid disease. Lafferty also thought the serum calcium was frequently greater than 14 mg/100 ml in patients with hypercalcemia of malignancy, while the serum chloride levels appeared to be lower and anemia was more common than in primary hyperparathyroidism. The response to adrenal steroids was greater in patients with malignancy, and he reported the serum phosphorus, serum uric acid, tubular reabsorption of phosphate, and calcium infusion tests were of little or no value. Differentiating between the two syndromes, Lafferty believed that the history of a rapidly progressive illness with marked weight loss and absence of a history of renal calculi, peptic ulcer and long standing skeletal complaints, favors the diagnosis of pseudohyperparathroidism. Ackerman and Winer[7] also stated that a history of hypercalcemia prior to the development of malignancy, as well as the type of tumor, were important in differentiating pseudohyperparathyroidism from primary hyperparathyroidism.

Many other papers have been published on laboratory tests to differentiate between the two syndromes.[8–10] Pointing out that many reports were available to explain the calcium abnormalities seen in various disorders, Plimpton[11] proposed that: (1) the serum calcium may be carried in unionized forms in some types of hypercalcemia; (2) tumors may produce substances which behave like extracts of parathyroid gland; (3) tumors may produce a vitamin D-like substance; or (4) there may be an abnormal substance binding the calcium and transporting it in excessive amounts. Each is difficult to prove and adds to the puzzle when approaching a patient with hypercalcemia. For instance, Powell[12] reported a patient who had pancreatic carcinoma and hypercalcemia in which parathyroid hormone antisera was used and no parathyroid hormone was found. He stated that almost one-half of the tumors in patients with pseudohyperparathyroidism have no detectable hormones. To explain the clinical and biochemical differences between patients with primary hyperparathyroidism and patients with pseudohyperparathyroidism, Powell stated that it is possible that a substance distinct from parathyroid hormone could have caused the hypercalcemia. The differences between these conditions would include acid base balance, suppressibility of hypercalcemia by means of corticosteroid therapy, and studies employing radioactive calcium and bone cultures.

In the past, clinicians stressed that the presence of bony metastases excludes the diagnosis of pseudohyperparathyroidism. Samaan[13] did not believe this was valid since in some instances treatment of localized metastatic disease resulted in disappearance of the hypercalcemia. He had also seen patients with hypercalcemia and bone metastases who became normocalcemic or tetanic after removal of the primary tumor only. In his M. D. Anderson series,[14] he gathered 78 patients over a two-year period who experienced an elevated serum calcium and elevated parathyroid hormone levels in neck veins. Primary hyperparathyroidism was confirmed at surgery in all these instances, although 25 had tumors in other parts of the body. Samaan believes that high levels of parathyroid hormone in the superior vena cava are not diagnostic. He also believes that the history of renal stones and radiographic evidence of subperiosteal bone

resorption is diagnostic of primary hyperparathyroidism, as is the finding of a high level of immunoreactive parathyroid hormone in the neck veins.

The case reported had evidence to support the production of a parathyroid-like substance by the islet cell tumor. Rasmussen[15] has demonstrated that parathyroid hormone is a single protein with a molecular weight of about 9,500. It is homogenous by countercurrent distribution, thin layer chromatography and ultracentrifugation, and contains no cystine. The single end terminal amino acid is alanine. Many smaller active polypeptides have been identified. In 1964, utilizing direct complement fixation, Tashjian[16] demonstrated a substance in urea extracts of six tumors that was immunologically similar to bovine parathyroid hormone extract. Arnaud[17] believes there is evidence of at least three circulating forms of immunoreactive parathyroid hormone in cases of primary hyperparathyroid disease. Riggs *et al.*[18] reported a smaller increase of parathyroid hormone serum level for an equivalent increase in calcium in cases of the ectopic syndrome than with primary hyperparathyroidism. Later studies demonstrated that a quantity of circulating inactive fragment of parathyroid hormone molecule (portion adjacent to the COOH terminal end) was greater in primary hyperparathyroidism than the ectopic syndrome. An NH2 terminal fragment (active fragment) was elevated in the ectopic parathyroid hormone-like syndrome. The assay used in the case presented was a radioimmunoassay using rabbit anti-bovine parathyroid hormone specific for the C-terminal fragment. Ectopic parathyroid hormone has been shown to be produced by a variety of tumors, most commonly oat cell carcinoma of the lung, hypernephroma, hepatoma, cancer of the pancreas, and colon carcinoma.[12]

Wiechert[19] demonstrated the neuroectodermal origin of peptide secreting endocrine glands. He proposed that peptide secretory endocrine tissue is related to the nervous system, not only by its coordination of bodily function, but also by development from primitive multipotential endocrine cells derived from neuroectoderm. Over 50 reported cases in which tumors have simultaneously produced not one but multiple peptide hormones support his theory, that the syndrome of multiple endocrine adenomatosis may simply be a dysplasia of neural ectoderm. Bronchogenic carcinoma, particularly oat cell, has been found to produce almost all of the peptide hormones, and these anaplastic carcinomas of the lung can also be related to the neuroectoderm. Friesen[20] further defined the endocrinopathies as functioning tumors in unexpected ectopic locations. Although some are situated entopically, as expected, they appear to function ectopically. Wiechert proposed and substantiated the concept that certain polypeptide hormone-producing cells of the pancreas (islet cells), thyroid gland, lung, adrenal gland, parathyroid gland and gastrointestinal mucosa, are derived from neuroectodermal cells. They migrate into the primitive alimentary mucosa, where they move passively to a definitive position where they mature. Neoplasia of such primitive neuroectodermal-derived cells inherits the potential to produce a variety of vasopressive amines and polypeptide hormones. This may depend on the functioning genes of these cells or derepression of other genes present in the cells. Perhaps changes in the differentiation of the neoplastic cells may be a mechanism of this repression.

Pearse[21] and others have demonstrated that most of the peptide secreting endocrine cells of the foregut have common cytologic characteristics in which the cells take up amines and decarboxylate them to form polypeptides (APUD cells). From their origin in the neural crest he demonstrated experimentally the migration of the APUD cells through the mesoderm to the foregut entoderm. He postulated that: (1) an arrest of this migration could result in ectopic mesodermal locations; (2) ectopic elaboration of hormone could occur if errant migration to an inappropriate site occurred; or (3) the precursor cells may have a multiple potentiality for humoral elaboration. Therefore, ectopia may be a result of error in embryologic migration and secretory potential. Friesen[20] suggested that cellular hyperplasia might also occur as a secondary manifestation in response to the environment of the cell. For example, a low blood sugar in a patient who had an insulin-secreting tumor may lead to hyperplasia of glucagon-secreting cells. This would explain multiple hormone production by a single tumor.

O'Neal[3] reviewed the literature relating to ectopic parathyroid hormone and found there has been a variety of hormonal substances produced by islet cell tumors. Assays of islet cell tumors were positive for ACTH, MSH, gastrin, and glucagon in the cases he reported. Clinical laboratory studies also suggested parathyroid hormone-like activity and vasopressin-like substances. It is believed that glucagon is secreted by alpha cells, insulin by beta cells, and gastrin by delta cells of the pancreatic islets. Frequently it is impossible to determine the cell type in islet cell tumors without sophisticated and often experimental methods. Insulin, glucagon, gastrin, ACTH, MSH, serotonin, possibly parathyroid hormone, ADH, and some substance producing watery diarrhea and hypokalemia have all been reported in association with islet cell tumors.[22]

Goldenberg,[23] studying the ultra-structural features of functioning alpha and beta cell tumors, showed a large range in size, shape, and degree of crystallization and a

number of secretory granules in these tumors. Suzuki[24] divided beta cell tumors into two major types: Type 1 showed the typical beta cell granules in the cytoplasm; Type 2 cells showed extensive interdigitation of their plasma membrane and contained few, mostly atypical, immature beta granules with some lysosomal elements. Metastasizing tumors were of the Type 1 variety. Other investigators[25-30] have documented occurrences of heterogenous groups of circulating abnormally large precursor polypeptide molecules in islet cell neoplasms. It is difficult to tell if one or all of the multiple hormones are contained in granules or if they are continually secreted or synthesized and then released in the blood stream. The case reported had the clinical findings indicative of hyperparathyroidism. It was reported as an islet cell tumor with some malignant characteristics, but no evidence of metastases. No definite cellular subtype could be determined and electron microscopy studies are pending. Furthermore, no explanation for the preoperative diabetes and amenorrhea can be given. Insulin assays were not obtained, and the assay used for estrogen was within normal limits. It can only be postulated that other hormone-like substances were secreted by this same tumor.

Summary

There have been many reports of pseudohyperparathyroidism with malignancy and many reports of ectopic elaboration of parathyroid hormone-like substances. However, reports of parathyroid hormone secreted by an islet cell tumor have been relatively rare. The parathyroid hormone-like substance may be found in several syndromes:[2] (1) hyperparathyroidism of entopic location such as seen with hyperplasia, adenoma, or carcinoma of the parathyroid glands with or without multiple endocrine adenopathies; (2) hyperparathyroidism of ectopic location arising from the APUD cell carcinomas of the foregut including the pancreatic islet cells; or (3) pseudohyperparathyroidism including some nonendocrine malignancies, such as the breast, lung, kidney, or ovary with or without metastasis of the bone. The more rare of these clinical entities and also most difficult to treat successfully are those of hyperparathyroidism due to malignancy of the parathyroid glands (entopic location) and of the pancreatic islets (ectopic location). Streptozotocin has been shown to be effective in islet cell tumors secreting parathyroid hormone.[1] This agent acts specifically on islet cells, and evidence suggests that the drug may be more effective in interrupting hormone production by the islet cell tumors than in causing cell death. It would be interesting to note the result of treatment with streptozotocin in our patient should a recurrence or metastases occur.

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Spider Angiomata

Treatment With Sclerosant Injections

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INJECTION SCLEROTHERAPY as treatment of varicose veins has been popular in the United States and abroad, but little work has been done toward the treatment of spider angiomata of the legs. In fact, little is known about the development of spider veins. Although they may appear concomitantly with varicose veins, they often develop during the teenage years and are independent of varicosities.[1]

From an anatomic standpoint spiders are intracutaneous, whereas varicose veins develop from the subcutaneous network of veins. Nevertheless, spiders are unsightly and of great cosmetic concern to many people. It was this concern which prompted the development of the new sclerotherapy technique of treatment.

Clinical Material

During an eight-year period, 286 legs (144 patients) have been treated with injections. All but one person were women. Ages ranged from 18-58 years, with an average age of 32 years.

All persons requesting treatment were accepted for therapy and are included in this report. Use of birth control pills, or a history of allergic or hypersensitive reactions, were not contraindications to treatment. No patients manifesting a serious systemic disease has presented for treatment, however.

Injection Technique

Of the sclerosant material available, one per cent sodium tetradecyl sulfate has been the most efficacious (Sotradecol, Elkins-Sinn, Inc.). Injections are made with a 3 ml disposable plastic syringe fitted with a 30 gauge needle. After filling the syringe with 1-1.5 ml of solution, it is held between thumb and third finger. The index finger depresses the plunger *(Figure 1)*. Skin preparation requires only swabbing with an alcohol-soaked sponge. The hand, resting on the skin, holds the syringe, needle bevel up, at approximately a 45-degree angle. The skin surface is pierced, and the sclerosant injected.

Experience is required to judge the force necessary to penetrate the vein. Usually, the vein is covered by little more than the epidermis. Hence, the needle may need penetrate no deeper than the bevel of its point. The skin prick itself is usually painless due to the minute size of the needle.

The sclerosant is injected with the index finger until the venous channels are filled and blood is momentarily expelled. This may require only a drop or two of fluid, or as much as 0.25 ml for larger vein complexes. The fluid is stroked into the periphery of the vein with alcohol swab. This is all that is necessary to treat the vein and takes but a few seconds for any one vein complex. The injections are continued in this manner, perhaps using 3-4 ml of solution and treating as many as 30-40 individual sites.

Utilization of injection sclerotherapy for treatment of spider angiomata of the legs is described. Results of the series reviewed indicate this to be a highly successful technique with virtually no adverse effects.

If the vein does not immediately fill, the needle is withdrawn slowly while pressure is maintained on the plunger. Occasionally, the vein will accept fluid during withdrawal if the needle has penetrated it.



Figure 1. A 30-gauge needle is fitted to a 3 ml disposable syringe for the injections.

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When the vein complex refuses to accept the sclerosant, the needle is withdrawn completely and the vein reinjected at a later time. Injections made outside the vein have little effect on the skin but will not cause obliteration. The fluid must be injected intravascularly for obliteration to occur.

Within a few minutes, a local allergic skin reaction develops at the injection site. This is evidenced by a typical "wheal and flare" which looks like an insect bite. Most people report that the injection feels like an insect bite as well. This local reaction may become quite pruritic and continues for about twelve hours. Although no special precautions or treatment are necessary, hot baths and astringent lotions help provide relief from the stinging, itching, and burning; ordinary exercise is encouraged. Patients are seen at weekly intervals and injections continued until the patient is satisfied with the number of veins treated.

A few days after injection, the larger venous channels may be seen as dark blue threads. This represents a small amount of trapped blood. Although it may be absorbed spontaneously, the process should be hastened by piercing the vein with a #23- or #25-gauge needle and expressing the old blood with the finger tips. If the blood is not completely removed, a brownish discoloration may develop along the course of the vein. This pigmentation is due to the deposit of hemosiderin. Although the venous channel itself is completely obliterated and invisible, the pigmentation may be apparent for many months until it is absorbed.

When the overlying epithelium of large spiders is extremely thin, it may be necrosed by the sclerosant and a small ulcer, covered with a shiny black scab, results. Healing progresses without incident, although a pale scar may remain.

Results

All patients have been followed by personal visit or telephone conversation. The longest follow-up time was 97 mos; the average was 50 mos.

There have been no major complications, either local or systemic. Most of the patients require four to six treatments over a period of two months. An average of 2.5 ml of material is injected at each visit to an average of 20 sites. One female had a total of 18 treatment visits over a 12-month period and required a total of 85 ml solution. Her skin showed no adverse or undesirable effects from the multiple, intracutaneous injections. Neither the number of injections nor the total quantity of injected sclerosant seems to adversely affect the surrounding skin.

All patients have been pleased with the cosmetic results of treatment. The only complaint is related to the prolonged length of time sometimes required for the resorption of the brown stain. This is usually less objectionable, however, than has been the appearance of the spider veins prior to treatment. Fortunately, pigmentation occurs in fewer than one-third of the legs. It is believed that prompt release of trapped blood will prevent most of the hemosiderin deposition and pigmentation.

It is known that snug wrapping of varicose veins will prevent thrombosis by collapsing the veins. Wrapping has not been used with the injection of spiders, but may well be useful when larger spiders are treated.[2]

Figure 2 shows a typical group of spider veins. In this patient, they are associated with varicose veins. The spiders were treated with a series of six injection treatments, using a total of 23 ml of one per cent Sotradecyl, over a period of seven weeks. The leg is shown 12 months later in *Figure 3*. All of the spiders have disappeared. The varicose veins remain since they were not treated. Some pigmented areas are still visible on the



Figure 2. The left leg shows typical groupings of spiders. They were injected with multiple injections of one per cent sodium tetradecyl sulfate over a period of seven weeks.

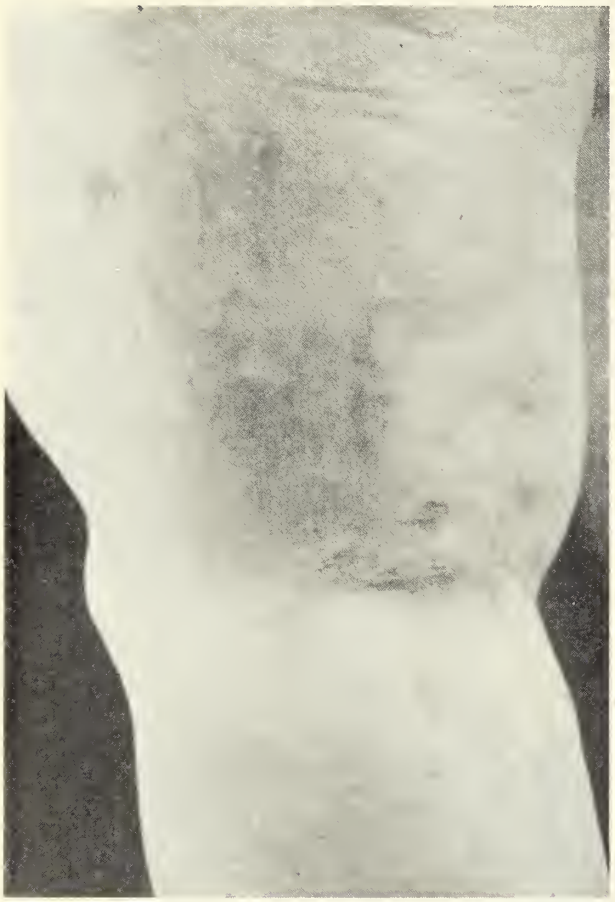


Figure 3. Spider veins have disappeared 12 months after treatment. Slight residual pigmentation of the skin can be seen where the spiders were large.

lateral thigh. The popliteal area is remarkably free of staining. Careful attention was paid to the release of trapped blood.

Although the treatment has not been offered to patients for the relief of symptoms in the legs, a number of patients have nevertheless reported subjective improvement of aches and pains. It is possible that larger complexes of spider veins may sequester enough blood to cause pressure symptoms just as larger varicose veins do.

Objectively, there are no recurrences of the veins treated. Many patients have returned for treatment of newly developed veins.

The injection sclerotherapy technique has proved to be a safe, relatively simple, and highly effective method of treating spider veins.

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Postoperative Ileus

Gastrointestinal Roentgenography During the Early Postoperative Period

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DESPITE extensive literature on the topic,[1, 2] present knowledge of postoperative gastrointestinal mobility and its control is far from complete,[3, 4] pathogenesis of the postoperative ileus is ill understood and surrounded by controversy,[5–10] and treatment of ileus is sometimes debatable[11, 12] or unsuccessful.[13, 14]

The postoperative gastrointestinal mobility has been evaluated by clinical procedures, balloon kimography, endoradiosondes, radioisotopes,[15, 16] electromyography[10] and radiological procedures using barium,[17] radiopaque spherules,[10] or hydrosoluble contrast media.[18] After more than half a century of work, it has been definitively demonstrated that the existing clinical impression of a global gastrointestinal paralysis after operation is not consistent with the facts. Moreover, not everything learned in the animal laboratory is applicable to the human species, thus defining the need for research with human volunteers.

The following report relates our experience with a group of human volunteers in whom the extent of ileus and its diverse components were quantitatively and comparatively evaluated in seeking the answer to the following three questions:

1. What is the effect of gastric delay, small bowel hypomobility and colon delay, upon the total duration of postoperative ileus?
2. To what extent does this type of operation, presence of peritonitis, and bowel manipulation affect the duration of ileus?
3. What degree of effectiveness can be expected from therapeutic measures promoting increased peristaltic small bowel activity?

Material and Methods

This study included 24 males and 11 females ranging in age from 20-75 years (mean 53 years). All underwent abdominal operation under general anesthesia. Postoperatively, the gastrointestinal transit time of a hydrosoluble contrast medium was monitored by serial abdominal radiographic pictures taken at intervals of one-half, one, two, three, four, six, eight, twelve, and twenty-four hours after contrast administration.

In 29 individuals, a mixture of 50 per cent sodium diatrizoate (Hypaque 50) 40 ml and saline 20 ml was instilled into the stomach through a Levin nasogastric tube, at variable intervals after operation. In 15 instances, the patient was immediately put in right lateral position for one hour. Subsequently, the patient was allowed to change position as desired. In the other 14 instances, the patient was allowed to remain in recumbent position throughout the first 2-24 postoperative hours. In either case, the nasogastric tube was clamped for one hour after contrast administration.

Results of a definitive study are presented in abrogation of traditional misconceptions regarding postoperative ileus.

Six of the patients who underwent cholecystectomy had intraoperative cholangiogram with Renografin 60, 20 ml. Timed radiographic pictures taken shortly before abdominal closure showed passage of the radiopaque dye into the duodenum in all instances. This allowed precise determination of the duration of the subsequent small bowel transit of the dye in these patients. To quantitate the effect of the gastric, small bowel and large bowel delay upon the total duration of the postoperative ileus, the following parameters were measured: (1) complete gastric retention time; (2) gastric emptying time; (3) small bowel transit time; and (4) total duration of the postoperative ileus.

Results

Gastric Delay. No side effects or complications resulting from sodium diatrizoate dye administration were detected. The dye accumulated in the gastric fundus of patients who were allowed to remain in recumbent posi-

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TABLE I
GASTRIC EMPTYING TIME

Time of Contrast Administration After Operations	*Gastric Delay*
A. 0-6 hrs	4 hrs 50 min ± 2 hrs 50 min
B. 6-24 hrs	2 hrs 15 min ± 3 hrs 2 min
C. 24-48 hrs	0 hr 48 min ± 0 hr 37 min

$F = 18.55479$ $F_{0.05} = 4.11$ $p < 0.001$ (ANOV)

TABLE II

Time of Contrast Administration After Operation	*Small Bowel Transit Time mean ± SD*
A. 0-6 hrs	6 hrs 30 min ± 1 hr 51 min
B. 6-24 hrs	5 hrs 10 min ± 3 hrs 34 min
C. 24-48 hrs	5 hrs 30 min ± 4 hrs 25 min

$F = 1.14$ $(F_{0.05} = 4.24)$ $p > 0.5$ NS

tion. It persisted there for 2 to over 24 hours, unless removed by gastric suction. Gastric emptying began within 30 min in patients who were kept one hour in right lateral position after contrast administration, except in those who underwent an operative procedure upon the gastric outlet (truncal vagotomy and pyloroplasty), who presented complete gastric retention for more than six hours. The gastric emptying time depended on when contrast had been administered. It was longest during the first six postoperative hours (4 hrs 50 min ± 2 hrs 50 min), and it significantly decreased ($p > 0.001$) when contrast was given during the second postoperative day, (0 hrs 48 min ± 37 min) *(Table I)*.

Small Bowel Transit Time. As shown in *Table II,* the length of the small bowel transit time depended also on the timing of contrast administration. Slightly longer transit times were found during the first six postoperative hours (6 hrs 30 min ± 1 hrs 51 min); however, there were no significant differences ($p > 0.5$) with transit times recorded during the remainder of the first postoperative day or on the second postoperative day.

Total Duration of the Postoperative Ileus. Whether the termination of postoperative ileus was defined as the instant in which the first passage of intestinal gas or the first defecation occurred, the total duration of the postoperative ileus was 2 days 10 hrs ± 1 day 5 hours, or 3 days 21 hrs ± 1 day 15 hrs respectively.



Figure 1. Visualization of the terminal ileum and ascending colon 7 hrs after partial intestinal plication (Noble operation) and 6 hrs after nasogastric Hypaque administration.

Abdominal radiographic pictures showed persistent dye in the colon throughout the observation period unless previously eliminated by defecation. No radiographic evidence of dye absorption (pyelogram, cystogram) was detected in any of the patients who received Hypaque. Two of five patients who received Renographin 60 had abdominal films showing cystograms 2 and 4 hrs after contrast administration.

The colonic transit time was assumed to be the time elapsed from the entry of dye into the colon until the first defecation occurred. Although this assumption introduced an element of uncertainty in our deductions, in five patients, who were followed with radiographic controls of up to five days, the dye in the colon disappeared after the first defecation in four instances and during a subsequent defecation six hours later in the fifth patient.

Effect of the Type of Disease and Operation Upon the Duration of Ileus. As expected, patients who had en-

TABLE III
DURATION OF ILEUS vs TYPE OF OPERATION

Type of Operation	*Duration of Ileus*
Cholecystectomy	3 days 5 hrs ± 18 hrs
Enterolysis or extensive bowel manipulation	4 days ± 1 day 2 hrs
Operation for peritonitis	3 days 14 hrs ± 2 days 14 hrs
Minimal bowel trauma	3 days 12 hrs ± 1 day 2 hrs
$p > 0.05$	NS (ANOV)

terolysis or extensive bowel manipulation had the longest postoperative ileus (4 ± 1 days), followed by patients with peritonitis (3½ ± 2½ days), while the shortest postoperative ileus was seen in patients who underwent little or no operative bowel manipulation (3½ ± 2 days). However, wide superposition of data and a wide range of variability within each series made these differences nonsignificant *(Table III)*.

Comments

Radiographic pictures obtained with Hypaque or with Renografin were of very similar quality, but while no absorption of Hypaque was radiologically detected, in two of the five patients who received Renografin, evidence of urinary elimination of the dye was seen. Urinary elimination of diatrizoate dye after oral administration has been interpreted in the past as a sign of loss of the normal gastrointestinal barrier (intestinal perforation, disruption of sutures, extensive ulcerations).[19, 20] Present findings suggest that investigation of such signs requires the use of sodium diatrizoate (Hypaque). Since intestinal absorption of Renografin (which also contains methylglucamine diatrizoate) occurs in spite of persistent normality of the gastrointestinal mucosa, interpretation of urinary elimination of dye is less accurate when Renografin is used. Intestinal absorption of other diatrizoate dyes, such as Gastrografin, has also been demonstrated.

Persistence of Hypaque in the stomach, when the patient was maintained in recumbent position, was not followed by any major side effect. Chemical changes (carboxylic acid formation) and physical changes (inspissation) have been reported by others[21] when gastrografin was left in contact with gastric juice for several hours. Pathologic changes (hyperemia, hemorrhage, necrosis) have been reported following administration of Gastrografin.[22-24] Changes were less marked with sodium diatrizoate without the addition of methylglucamine and detergent, both present in Gastrografin.

Positional changes favored the gastric evacuation except in patients who underwent pyloroplasty and truncal vagotomy, possibly because postoperative pyloric edema interferred with gastric evacuation during the first 48 postoperative hours.[25] The analysis of the gastric emptying time showed a remarkable delay in operated subjects when compared with generally accepted emptying times in non-operated individuals, a finding consistent with prior experience with operated patients[26] and animal laboratory studies.[5] Dogs who swallowed radiopaque spherules increased their preoperative gastric emptying time from 1 hr 25 min to 65 hrs postoperatively.[10] Less dramatic increases were noticed in other animals. The extent of intestinal injury or manipulation increased the gastric delay in experimental animals.[16] In our series, the delay in gastric emptying was largest immediately after the operative trauma (4 hrs 50 min) and decreased thereafter throughout the 48-hr observation period. However, while there was a continuously decreasing trend, a significant decrease was noticed only after the first 24 postoperative hours. These results showed that the gastric postoperative ileus, although intense, lasted barely beyond the first 24-hr period in this series ($p > 0.0001$), and even during the first six postoperative hours, the gastric ileus accounted only for 3-7 hrs of the total ileus time in these patients.

The postoperative behavior of the small bowel has been the object of controversy. It is now admitted that participation of the small bowel hypomotility in the total duration of the postoperative ileus has been largely overemphasized.[3-5] Some more recent data suggest that the small bowel motility may be undisturbed postoperatively.[25] The auscultatory silence is due to the absence of bowel contents,[4] and vigorous contractions occur, even in presence of sepsis, peritonitis, or pelvic abscesses. The electric intestinal activity pattern has been found undisturbed in fasted dogs after abdominal surgery,[4] but decreased for four days in human patients.[2] Others found that while celiotomy did not disturb it, extensive manipulation decreased small bowel motility.[5] Our present results corroborate a decreased small bowel motility after operation, persisting with slight changes throughout the observation period. Normally, it takes 45 min-3 hrs for Hypaque to travel from the pylorus to the ileocecal valve.[27] The transit time was more than triple this value after the operation. Transit times of up to 9 hrs were recorded in some patients without proven mechanical obstruction. The importance of these findings relate to the fact that Hypaque gastrointestinal studies have been suggested for differential diagnosis of mechanical obstruction and ileus. The record in this study of longer transit times than those generally accepted emphasizes that while a transit time of less than 4 hrs eliminates the possibility of postoperative mechanical obstruction, longer

transit times do not necessarily mean that a mechanical obstruction is present.

Due to dilution of Hypaque by the succus entericus, visualization of the mid-gut was somewhat unsatisfactory. Reconstruction of contrast medium in the ileum and colon allowed for adequate visualization of these intestinal segments.

The colon transit time—calculated from the passage of dye through the ileocecal valve until contrast elimination via rectum occurred (first defecation)—showed tremendous variations. The situation was further compounded by the extreme variability of the colon transit in non-operated subjects.[28] Individual analysis of each transit time showed that in all instances the colon delay was the major component of the total duration of the postoperative ileus, while the gastric and small bowel delays, although present, were too small to have had any significant effect on the duration of ileus. Consequently, any therapy aimed at reducing the duration of ileus should be directed toward a reduction of a colic delay.

As orginally stated, therapy for postopertive ileus is controversial and results are less than satisfactory in many instances. It is hoped that the impact of the colic delay upon the total duration of ileus will be taken into consideration when new therapeutic measures to decrease the duration of postoperative ileus are designed.

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Augmentation Mammaplasty

Fibrous Capsular Contracture and Other Complications

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AUGMENTATION MAMMAPLASTY is a cosmetic operation performed to fulfill the patient's desire for normal appearing breasts. During the past decade or so, it has gained in popularity in our American society and is now just as commonplace as such other cosmetic operations as rhinoplasty, facelift, and removal of eyelid wrinkles. The technique involves the insertion of a foreign body into a surgically-created pocket between the pectoral fascia and the breast parenchyma.

Early methods of augmenting the breast are of historical interest only. First, there were the autogenous tissue transplants which used free fat grafts from the buttocks; these were not successful because they underwent absorption. Dermal-fat pedicle flaps from the inframammary regions were an improvement, but it was difficult to achieve good symmetry and the tissue obtainable frequently did not provide sufficient bulk. Next came the sponge-like prostheses made of various synthetic materials (Ivalon, Teflon, Etheron, and others); they furnished good form and a selection of sizes, but had their drawbacks. Infections were more frequent and they invariably became infiltrated by the ingrowth of fibrous tissue—the sponges metamorphosed into rocks! The silicone injections were utter disasters, achieved notoriety, and were quickly banned by the FDA. It is incredible but true that certain unscrupulous persons continue to use this method.

The above-mentioned techniques have been supplanted by the silicone formed prostheses which are membrane-like structures containing saline or silicone in the gel state. Technological refinements have given this type of prosthesis a softer, more natural feel by making the membrane thinner and the gel less viscous. A vast array of sizes and shapes is available on the market—the inevitable result of competition between the now half-dozen or more manufacturers (Dow Corning Corporation is the Coca-Cola of the industry).

The incisional approaches are the infra-mammary, the peri-areolar and the trans-axillary, diminishing in that order as the preferred method by various surgeons. This type of surgery is no different from any other—things can go wrong.

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Complications

Acute hematoma

An acute swelling of the breast accompanied by an undue amount of postoperative pain within the first 12 hours makes the diagnosis obvious. This can occur even though wound suction is employed. If it does, the most effective treatment is to return the patient to the operating room, prepare her in the same manner as for the initial surgery, and evacuate the blood clots (seldom is an active bleeding point discovered). The prosthesis is replaced and the wound closed as before. In our experience this procedure has been necessary in only five of approximately 400 breasts. We routinely use Hemovac drains as a prophylactic measure because of the impressive total amount of drainage over a period of 3-4 days after the operation. When the danger of acute hematoma has passed—usually in two days—the patient is released and allowed to manage her Hemovac at home, thus moderating the hospital expense.

The history of cosmetic breast augmentation is reviewed and the present procedure outlined. Prevention and treatment of complications are discussed.

Infection

This is a rare complication that usually responds to conservative measures. Most infections originate in the operative incision and this localized site allows easy accessibility for treatment. In twelve years we have encountered few infections and have never had to remove an implant because of one. Prophylactic antibiotics are not necessary. Our experience indicates that the above two complications have a lower frequency than one might expect.

Numbness

This condition is usually of a transient nature in the lower portion of the breast, but occasionally it may involve the nipple-areolar complex, in which case it is permanent.

Asymmetry

This is a possibility, but occurs infrequently. The sur-

gical pocket does not have to accurately fit the implant (although care must be taken not to make it too low); in fact, it should be considerably larger for reasons that will be mentioned later.

Ptosis

Only the most minor degrees of ptosis can be eliminated by this method; it usually requires a more elaborate mastopexy procedure. The unwary surgeon seeking to correct this condition by the insertion of a prosthesis is very apt to increase its severity rather than correct it.

Fibrous capsules

This term refers to the envelopment of the implant in a progressively contractile scar which gradually becomes more spherical in shape. It is far more common than any of the previously mentioned complications. The compression of the prosthesis into a smaller space gives it an unnatural firmness which can vary considerably from one patient to another. If it is sufficiently pronounced it can lend a globular appearance to the breast. The extreme case may resemble the hard rubbery consistency of citrus fruit, and is often accompanied by pain. Such contracture can develop up to as late as 24 months.

Causes

The precise cause of fibrous contracture still remains obscure and it is probable that there are multiple factors at work. Some have thought that subclinical hematoma formation is the culprit; controlled animal experiments have shown that a thicker capsule develops around an implanted piece of silastic with blood injected into the cavity through a catheter following wound closure. For this reason, many surgeons routinely utilize postoperative wound suction, but there is no proof that their results are better. Others have experimented with one to three weeks of postoperative immobilization to reduce bleeding resulting from movement of the pectoral muscles. Unilateral involvement is more common than bilateral, but case anlysis has failed to show correlation between the hand dominance of the individual and the side of contracture development. No objective criteria have been established for valid comparison of results of these procedures, leaving the surgeon to rely primarily on his own subjective impressions.

Electron microscopy recently has shown the presence of vacuoles within the wall of the capsule, and these are surrounded by a cellular infiltrate consisting of mononuclear cells, macrophages, giant cells, and the so-called contractile or myofibroblast. The silicone membrane of the prosthesis is known to be semipermeable and it appears that these microscopic entities are indeed dimethylpolysiloxane, *i.e.*, silicone. To explain capsule formation when the saline inflatables are used, it has been postulated that a shedding phenomenon occurs, liberating particles from the membrane surface.

Wound healing is still imperfectly understood and the existence of extremely complex biologic and biochemical processes may be responsible in part for the tremendous variation in the amount and character of the scar tissue formed from one individual to the next. In the face of this, it is problematical how much surgical technique is able to influence the end result.

Prevention and Treatment

Mention has already been made of the possible role of hematoma formation as related to the amount of capsular response and the methods employed to avert its occurrence, *i.e.*, attention to hemostasis, postoperative restriction of activity, and the use of wound suction. Triamcinolone (Kenalog and Aristocort) is one of the accepted treatment modalities for cutaneous scars, but its effect in softening hypertrophic scars is unpredictable. Because of this visible and palpable effect on external scars, some surgeons make a practice of instilling it into the dissected cavity at the time of the original augmentation procedure, not only at re-operation (see below). The material is injected through a catheter just prior to wound closure in an effort to distribute it evenly. A serious drawback, however, is that triamcinolone can cause atrophy of the subcutaneous fat leading to a thin skin cover (and occasionally actual erosion) in the lower portion of the breast where it tends to accumulate from the effect of gravity. Therefore, some surgeons feel that this hazard does not warrant its use.

A few years ago it was believed that it was essential to have the prosthesis fixed to the chest wall to prevent lateral displacement as well as ptosis. Dacron mesh "fixation patches" affixed to the posterior surfaces of the implants allowed fibrous tissue to form an adherent bond. Gradually it was discovered that this fixation was not in the least necessary, and it was theorized that the patches might contribute to the formation of a thicker capsule.

The creation of over-sized surgical pockets, that is, larger than the prosthesis itself, seems to have definite value. By applying proper manual pressure the surgeon begins early—a few days postoperative—manipulation of the prosthesis in order to maintain the size of the space as much as possible. The patient is instructed to carry on this procedure during the ensuing months. In this way the capsule may be kept at a distance, so to speak, and when some contracture eventually occurs it does not press so directly upon the implant; it may also act to keep the capsule constantly attenuated so that it is

(Continued on page 217)

Deep Venous Thrombosis

Methods of Laboratory Diagnosis

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UNRELIABILITY of clinical diagnosis of deep venous thrombosis (DVT) has been repeatedly documented in the medical literature.[1] Isolated soleal vein thrombosis may escape clinical detection. While this is generally inconsequential, 20 per cent of these DVT propagate centripetally, and about 50 per cent develop pulmonary embolism without ever having had premonitory physical findings or symptoms.[2] Moreover, lower extremities with some classical physical findings (swelling, pain, color, temperature changes) failed to have phlebographically demonstrable DVT in almost 50 per cent of instances.[1]

Short-term anticoagulation therapy resulted in a 15-per cent incidence of bleeding episodes requiring more than two units of blood transfusion; the mortality rate was 0.6 per cent.[3] There is the risk of a possibly lethal pulmonary embolism in patients with DVT who are denied anticoagulation therapy; on the other hand, such therapy, with its inherent risks, should not be administered to patients not positively diagnosed with DVT.

The laboratory diagnosis of DVT was intended to correct these shortcomings. It was not intended to substitute for clinicial acumen, but to improve its accuracy. Over the past 30 years, many invasive and non-invasive diagnostic methods were enthusiastically presented as the ultimate solution of the problem. Further evaluation showed that each of these tests, while having positive aspects, also had severe limitations which decreased its applicability.

Presently there is no test that is totally sensitive or reliable. None of them can be used in all patients and some of them, although ingenious research tools, have little or no application to daily patient care. During the past five years, numerous research projects have addressed the problem of screening large populations at high risk for DVT. Currently there are authors who strongly believe that it is more desirable to stress prophylaxis of DVT in these patients than to subject them to serial cumbersome and time-consuming screening tests which are not 100 per cent accurate.[4] Excellent results of low dose heparin prophylaxis at subtherapeutic levels partially support such contentions.

Concepts in diagnosis of DVT are changing rapidly, and new diagnostic modalities emerge every year, some of which become obsolete before large scale trials have been performed. This paper will examine methods presently utilized by the Vascular Laboratory of the Truman Medical Center, Kansas City, Missouri, outlining those that best complement clinical diagnosis in the following categories of patients:

1. patients with presumptive clinical diagnosis of DVT;
2. patients with longstanding venous obstruction and suspicion of active phlebitis; and
3. patients on anticoagulation therapy in whom therapeutic failure (propagating thrombosis) is likely to occur.

Various diagnostic procedures are analyzed and the indications for utilization of each are discussed.

Presumptive Clinical Diagnosis

A definitive diagnostic method with great sensitivity and reliability is required to corroborate the clinical suspicion. Available methods detect either the distorted venous anatomy or the impaired venous function, with the anatomic methods being the preferred ones.

Radiographic phlebography, the golden standard of diagnosis for three decades, is still being used but less frequently than in the past. A six per cent incidence of thrombosis in previously normal veins has been reported following its use,[5] and immediate or delayed pain occurred in more than 50 per cent of all subjects. Allergy, skin slough, venous gangrene, syncopal episodes, and death have also been reported after phlebography. It is a cumbersome and time consuming technique which must be performed by highly skilled individuals; results do not always justify the time, expenses, and patient suffering involved in its use.

Improper tourniquet application, insufficient contrast

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medium injection, and venous collapse due to increased subfascial pressure often result in a false positive diagnosis. Conversely, thromboses in the profunda femoris or hypogastric veins are frequently missed by phlebography. In spite of these shortcomings, phlebography is still used, because none of the other techniques have proved superior. Contraindications and limitations of radiographic phlebography have resulted in the use of other tests in some instances.

Radionuclide phlebography is also an anatomic method and utilizes intravenous injection of radiopharmaceutical instead of the irritative radiological contrast medium. Radionuclide angiography is better tolerated, because no appreciable endothelial damage is produced and radiopharmaceutical extravasation was not accompanied by significant pain or local reaction. With the exception of very rare cases of idiosyncrasy, no adverse reactions have been reported following its use. By recording the data on computer videotape, subsequent analysis was possible, allowing evaluation of both the distorted anatomy and the impaired venous function. If Technetium-99m macroaggregate of albumin (Tc 99m MAA) was used to perform the radionuclide angiography, Tc 99m MAA ("hot spot") venous scan and perfusion lung scan could also be obtained without new radiopharmaceutical injection.[6] Radionuclide angiography improved by Tc 99m MAA venous scanning results in directly showing the venous clot as a suspended area of radioisotope activity several minutes after the radiopharmaceutical cleared from the blood stream. It could be performed in patients allergic to the radiographic contrast media and in those with renal failure, where diatrizoate contrast media injection was contraindicated. However, anatomic determination of images resulted in a less accurate diagnosis than radiographic phlebography, while profunda femoris and hypogastric veins could not be visualized with this method. As with radiographic phlebography, radionuclide phlebography cannot be performed in patients who do not have foot veins suitable for intravenous injection. The use of a tourniquet and leg exercising are recommended with both methods, but cannot always be performed because of pain and patient uncooperativeness. Since radionuclide phlebography and scanning does not require patient preparation or waiting period, these tests are suitable for emergency diagnosis of DVT.

The fibrinogen uptake test (FUT) is rarely indicated for initial diagnosis of DVT in clinical practice, primarily because patients with DVT cannot afford to wait at least 24 hours for serial FUT to be performed before initiating treatment. FUT can be used advantageously when exact assessment of DVT limited to the calf segment is desired.

Non-invasive methods, such as Doppler ultrasound venous flow detection, strain gauge plethysmography and volume plethysmography, in spite of enthusiastic reports of their proponents, have not achieved an accuracy comparable to phlebography.

Non-invasive methods for detection of venous thrombosis should be performed in pregnant women and children in whom radionuclide or radiographic methods may be contraindicated.[7] Non-invasive detection has also been useful in patients without veins suitable for injection, allergic patients, and in those refusing more invasive testing.

Doppler ultrasound venous flow detection is best performed with the patient in semi-Fowler position with slight knee flexion. A detector probe with a frequency of 8 MHz or higher has been advised to eliminate contamination of signals coming from the tested vein by signals coming from deeper venous structures, resulting in falsely normal findings.[8] The sensitivity of Doppler ultrasound venous flow detection for diagnosis of DVT has varied from 31-93 per cent, and its specificity from 41-93 per cent according to several authors.[9-11] Since a negative finding (absence of detectable venous flow) is considered diagnostic of venous obstruction, the incidence of falsely normal results depends to a great degree on the experience of the person performing the test, and on the manner in which the test is performed. False normal results may be due to improper technique or presence of large patent venous collaterals close to an obstructed vein.

Plethysmographic techniques and their variants, including electric impedance plethysmography and phleborreography, were 96 per cent accurate in detecting DVT. According to other investigators, the sensitivity of other plethysmographic techniques varied from 31-97 per cent, and their specificity varied from 16-97 per cent.[12-14] Plethysmography cannot be performed in patients wearing lower extremity casts (who indeed are high risk candidates for development of DVT), or in those in shock or with congestive heart failure. Patient cooperativeness is required. Tremor proscribes test performance. Pneumatic cuff inflation is used to momentarily suppress the venous outflow from the segment of limb to be evaluated. Nonlethal pulmonary embolism has been suspected in at least one instance immediately following cuff inflation to a pressure of 75 mm hg,[15] thus raising a question regarding the safety of the method for detection of acute DVT. Mural nonobstructing thrombosis of large veins may not be detected by plethysmographic evaluation. Moreover complete large vessel obstruction may also be missed in instances of extraordinary development of collateral venous circulation because plethysmography, a functional method, showed impairment of the entire venous outflow, but not the in-

dividual anatomic lesions. As with the preceding methods, plethysmography will not detect femoral profunda or hypogastric vein obstruction.

Detection in Patients With Past History

Some patients with postphlebitic syndrome had, in our experience, recurring bouts of thrombophlebitis which progressively worsened their clinical situation. While some of these instances were recognized beyond doubt, questionable clinical findings in others suggested the need for further evaluation. Radiographic phlebography did not unequivocally differentiate active phlebitis from longstanding obstruction in these patients, except under very favorable circumstances. Moreover phlebography (a potentially thrombogenic procedure) was of questionable value in these patients due to their low venous functional reserve. Serial FUT proved to be the method of choice for detection of active phlebitis in patients with past history of venous obstruction.[16] When serially increased per cent isotopic uptake was detected,[17] a methodic search for extravascular fibrin deposits was undertaken. If these could be ruled out, it was concluded that intravascular thrombi were actively developing and anticoagulation therapy was offered to these patients.

Therapy Failures

Prior experience in our laboratory[18] showed that about 33 per cent of all DVT patients on anticoagulation therapy exhibited evidence of propagating thrombosis. Extension of thrombosis occurred even in presence of standard laboratory test results (partial thromboplastin time or activated clotting time) showing full or excessive anticoagulation. Massive pulmonary embolism occurred in a patient who was concomitantly bleeding due to excessive anticoagulation therapy.[19] As previously shown,[17] anticoagulation failures were detected by serial FUT, by showing evidence of propagating thrombosis in the absence of clinical signs and before massive pulmonary embolism occurred.

FUT was not practical in patients with extensive lower extremity burns, ulcers, or eczema. Hematomas, wounds, and infection also interferred with data collection. When prolonged testing was needed, new injections of the radiopharmaceutical substance were necessary in most patients, because with use of commercial preparations of radioiodinated fibrinogen only 30 per cent of all injected material was still circulating five days after injection.[20] Degraded and denatured material contained in some of these preparations had decreased affinity for thrombi.[21]

To extend the benefits of serial monitoring to anticoagulated patients in whom FUT testing was impractical, we evaluated the results of serial Doppler ultrasound flowmetry in a series of 10 anticoagulated patients. There were two incidences of massive embolism. Propagating thrombosis was detected in both instances by the Doppler method before clinical debut of pulmonary embolism.[19] Best results with serial Doppler ultrasound monitoring were obtained when findings at the level of the dorsalis pedis, posterior tibialis, popliteal, superficial, and common femoral vein were investigated at least daily, and carefully recorded for serial correlation. Since qualitative changes were investigated, it was essential that all testing be made by the same person, preferably a physician, using a standardized technique and always the same equipment. Although our preliminary results with Doppler were encouraging, we believe that FUT will be ultimately shown to be superior to the Doppler method in the detection of propagating thrombosis, and consequently should be utilized whenever possible. Results of ongoing comparative analyses in our institution may help to delineate the relative value of both methods.

Summary

In spite of its well documented imperfections, radiographic phlebography is still the method of choice for emergency and definitive diagnosis of deep venous thrombosis. Radionuclide phlebography combined with hot-spot venous scan is chosen when phlebography is contraindicated or impractical. Non-invasive methods are less reliable and should be used for definitive diagnosis only when other more exact methods cannot be utilized.

Patients with longstanding history of DVT and a questionable bout of acute phlebitis should be evaluated with FUT. Patients with DVT on anticoagulation therapy should be monitored with the classic laboratory tests (ACT, PTT) and also with serial FUT, to detect failures of anticoagulation therapy and thus to enable initiation of therapeutic changes before occurrence of massive potentially lethal pulmonary embolism.

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The APUD Concept

(Continued from page 193)

pancreatic islets, the normal parathyroid hormone molecule from the normal parathyroid glands will be normal or suppressed by the high levels of serum calcium; the assay is measuring parathyrin from the normal parathyroid gland and not that from the ectopic pancreatic tumor. Special assays developed to recognize the larger heterogenous molecule of the polypeptide having parathyrin-like activity are necessary to recognize the humoral culprit in ectopic hypercalcemia or ectopic hyperparathyroidism.

The APUD concept and the theory of derepression thus explain most features of ectopic endocrinopathy.

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Abdominal Gunshot Wounds

A Review From Low Volume Trauma Centers

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MAJOR TRAUMA CENTERS have provided data for several published papers on gunshot wounds (GSW) of the abdomen.[1-8] However, we are aware of no reports from hospitals seeing a low volume of such cases. This paper presents the results of GSW of the abdomen from community hospitals dealing with a smaller number of cases, and compares these results with those from major trauma centers.

Subject and Methods

This is a retrospective study of GSW of the abdomen in patients subsequently undergoing exploratory laparotomy in three private hospitals in Wichita, Kansas. Wichita is a community with a metropolitan population of 305,000 situated in the south-central portion of Kansas.[9] The three community hospitals whose charts were reviewed are: St. Francis Hospital (SFH), staffed with surgical residents during the entire period; St. Joseph Hospital (SJH), with no surgical residents; and Wesley Medical Center (WMC), with surgical residents from 1970 to the present time. The bed capacities of these hospitals, excluding bassinettes, are 886, 439, and 717 respectively, totalling 2,042 (87%) of the city's 2,361 hospital beds.[10] The number of cases provided by each hospital for this review includes SFH—31; WMC—17; and SJH—7.

The management of the cases reported was different from that in most major trauma centers. Private physicians or residents working for a salary staffed the emergency rooms. Many of these were non-surgeons. After evaluation and treatment initiated by the emergency room physician, a private surgeon or surgical resident on call was notified to assist in resuscitation and assume management of the patient. There is no triage system or specific protocol for emergency room handling of GSW of the abdomen in these emergency rooms. A total of 23 surgeons were involved in the 55 cases, with a range of one to six cases each.

The medical records department in each hospital utilizes computerized records systems, and all charts coded as "GSW of the Abdomen" were reviewed. Superficial injuries and patients not undergoing exploratory laparotomy were not included. A protocol sheet was developed and utilized by each of the authors to obtain data from each chart reviewed.

Results

Males accounted for 48 cases (87%), 25 white and 23 black. White females made up the remaining seven cases (13%). The age range was 13-72 years with an average age of 34 years. Overall, there were six (11%) negative laparotomies and a mortality of 12 patients (22%).

Fifty-five cases of gunshot wounds of the abdomen from three community hospitals handling a low volume of this injury are reviewed. Information for fatalities and survivors is presented, and comparisons are made to results from major trauma centers.

Shock on admission was defined by the reviewers as a systolic blood pressure less than 80 associated with heart rate of over 100 beats/min. Of 43 patients, 13 (30%) in shock on admission survived, while 10 of 12 (83%) of fatal cases were in shock on admission. Overall, 23 of 55 (40%) of the cases reviewed were in shock on admission; 10 of these 23 (43%) died.

Time from the emergency room to the operating room ranged from 30-729 minutes (113 minutes average) for patients surviving, and 5-105 minutes (49 minutes average) for fatalities. Patients arriving in the emergency room in shock were in the operating room in an average of 98 minutes, while those not in shock in 79 minutes.

Hospital stay averaged 22 days with a range of 4-112 days. One patient deserted on the fourth postoperative day and was lost to follow-up. The majority of the hospital stay of the patient hospitalized 112 days was for psychiatric treatment. Without these two patients the average hospitalization period was 20 days.

Tables I and *II* analyze organ injuries and cause of death in the fatal cases. Information was included only

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TABLE I

Case	Number of Organs	Major Organs Injured
1	3	Duodenum, colon, kidney.
2	8	Diaphragm, duodenum, gallbladder, kidney, liver, pancreas (body), small bowel, stomach, lung.
3	4	Duodenum, liver, pancreas (body), stomach.
4	6	Diaphragm, liver, pancreas (body), small bowel, spleen, large vessel (inferior vena cava).
5	4	Kidney, pancreas (tail), spleen, ureter.
6	3	Diaphragm, spleen, stomach, shattered iliac crest.
7	4	Colon, liver, small bowel, stomach.
8	5	Colon, kidney, pancreas (tail), spleen, stomach.
9	2	Colon, large vessel (hypogastric artery and vein).
10	5	Diaphragm, colon, kidney, pancreas (body), spleen.
11	5	Colon, mesentery, small bowel, ureter, large vessel (right common iliac vein, inferior vena cava).
12	4	Colon, mesentery, small bowel, large vessel (abdominal aorta, inferior vena cava).

for patients who died as a direct result of GSW of the abdomen or a complication thereof.

Data gathered from the charts of surviving patients is summarized in *Tables III, IV* and *V*. *Tables IV* and *V* exclude negative laparotomies.

TABLE II

Case	Cause of Death	Time Post Admission
1	Colostomy breakdown, subdiaphragmatic abscess, sepsis	Day 32
2	Hemorrhage	Day 1
3	Acid/base imbalance, shock	Day 14
4	Hemorrhage	Day 1
5	Hemorrhage	Day 1
6*	Hemorrhage	Day 1
7	Sepsis	Day 7
8	Hemorrhage	Day 1
9	Sepsis	Day 18
10*	Hemorrhage	Day 1
11*	Hemorrhage	Day 1
12*	Hemorrhage	Day 1

* Cardiac arrest on operating table

TABLE III

NUMBER OF ORGANS INJURED IN SURVIVING PATIENTS

Organ Injuries	Number of Patients
0	6 (14%)
1	12 (28%)
2	9 (21%)
3	10 (23%)
4	5 (12%)
5 or more	1 (2%)
	43 (100%)

TABLE IV

SPECIFIC ORGAN INJURIES FOR SURVIVING PATIENTS

Organ	No. Patients	% Cases Involved
Small bowel	17	46
Liver	12	32
Colon	11	30
Stomach	10	27
Pancreas	7	19
Spleen	7	19
Kidney	4	11
Large vessel	3	8
Duodenum	1	3
Bladder	1	3

TABLE V

MAJOR COMPLICATIONS AS A DIRECT RESULT OF GSW OF THE ABDOMEN

Complication	Number
Sepsis	8
Pancreatitis	2
Severe atelectasis	2
Congestive heart failure	1
Erosive gastritis	1
Abdominal abscess	1
Fistula:	
enterocutaneous	1
pancreatic	1
Nerve injury:	
left foot drop	1
Urethral stenosis	1
Delirium tremens	1
Psychiatric	1
	22

Comments

This is a retrospective study of data from three community hospitals handling a low volume of gunshot wounds of the abdomen. We believe that statistical conclusions are not justified from our small series. The main purpose of our study is to present data and to compare our results with those of larger series.

Incidence. There appears to be an increasing incidence of gunshot wound of the abdomen in recent years. Most of the injuries were from handguns. Information gathered from charts in the first part of 1977, but not included in this study, indicates that the incidence of gunshot wound of the abdomen continues to be 10-12 cases/year in our community. Negro males made up a disproportionate number of these cases. Age distribution is consistent wtih that found by others reporting larger series.[2, 4, 7]

Shock. Of patients who subsequently died, 83 per cent were in shock on admission; however, an almost equal number of patients arriving in the emergency room in shock survived. This would indicate that shock on admission alone is not a good predictor of survival. When used in conjunction with the amount of fluid or blood required in resuscitation, or with the finding of major vessel injuries at operation, this becomes more significant. Overall, 43 per cent of patients in shock on admission died. This is similar to results reported by Nance,[4] who emphasizes that shock should be a mandatory indication for early exploration.

Organ Injuries. All but three of the fatalities had four or more organ injuries. Of patients who survived, 14 per cent had four or more organ injuries. The distribution of organ injuries correlates well with that of larger series.[3, 4] Colon injuries were found in seven, and large vessel injuries in four of the twelve fatalities. There were six negative laparotomies (11%). This incidence has been noted in several larger series.[2, 4, 7]

Cause of Death. There were no deaths in six laparotomies with no organ injuries. Hemorrhage was the most common cause of death, and sepsis the most frequent cause of late death. Colon penetrations were found in all cases of fatal sepsis. Overall mortality was 22 per cent. Davisson[2] and Taylor[7] report mortality rates in their larger series to be 10 per cent and 12.7 per cent respectively. Immediate mortality (within the first 24 hours) was 15 per cent; four patients died with severe shock and cardiac arrhyhmias in the operating room. Three of these had five or more organ injuries. Of fatalities, 83 per cent were in shock on admission. Nance,[4] in his series, reported a mortality rate of 42 per cent among the patients with five or more organs injured.

Complications. Complications such as minor urinary tract infections were not included in the table of major complications. Two entries—delirium tremens and psychiatric complications—were not a direct result of gunshot wound but were coexisting factors. The complication rate in survivors was 22 in 43 cases. Davisson[2] reported a complication rate of about 50 per cent in his series, while Nance[4] reported a rate of 36 per cent.

Summary

There are several differences in the management of GSW of the abdomen in community hospitals compared to major trauma centers: (1) the total number of cases is small; (2) the number of such cases handled by any individual surgeon is small; (3) there is no triage system; and (4) there exists no specific protocol for the emergency room management of the patient presenting with a GSW of the abdomen. Nance[4] promotes a policy of selective observation in the management of stab wounds of the abdomen. He further states that this policy may spare some patients with GSW of the abdomen with no intraperitoneal injury an unnecessary laparotomy. However, such a policy may not be practical in a center where a low volume of GSW of the abdomen is seen. Operating facilities in the emergency room have also been suggested as a means to further salvage a small number of fatal cases.[7] The effectiveness of this in a center where a low volume of trauma is handled might be questioned.

Demographic data, shock, organ injuries, mortality, and complications were the main points of comparison with major trauma centers used in our series. Based on these results, it appears that the record of cases in this series of GSW of the abdomen is comparable to that of major trauma centers.

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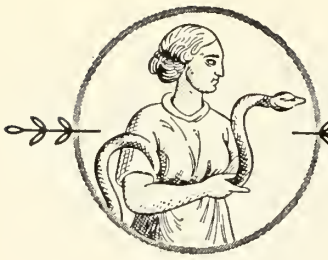
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Medical HISTORY

Back to Basics

*As Illustrated by Famous and Infamous Medicine Men**

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MANY HAVE EXPRESSED the concern how very difficult it is these days to practice medicine as we would like. I share these feelings as recurrent frustrations mixed among intervals of satisfaction. It is good to occasionally take stock in this day and age, when more distractions seem to demand so much of our time and divert us from whatever noble goals we once set for ourselves.

Recently, while attending a meeting of the International Society of Surgery in Kyoto, I listened to the welcoming address of the host and honorary chairman which, though given in lyrical Japanese, was of apparent emotion and warmth.[1] A translation revealed the following excerpt from his speech: "I would like to speak about the morality of the doctor. It is very regrettable that the medical profession is said to have degenerated these days. . . . There is an old German saying, *'Der Chirurg muss in erster Linie Wissenshaftler, in zweiter Linie Künstler, in dritter Linie, aber nicht in letzter Linie, Mensch sein,'* and another from the Chinese saying meaning, 'Do not hesitate to admit and set right when you make a mistake.' . . . I hope all the participants of this Congress will not only exchange information about medical affairs, but also communicate with each other on human affairs." His obvious sincerity set me to wondering if the suspect disintegration of morality were not more apparent than real, evident mostly because of increased pressures of society on surgeons already harassed by extra work. This increasing volume of paperwork and committee activity has surely taken time away from that which we might spend in a more professional way. Just as it is the recurrent call of the educators these days, we too must somehow get back to the basics.

These thoughts prompted a second look at the lives of some others who in their own way illustrate a way of getting back to the basics. Without getting overly serious about the subject, I was struck by the diversity of their deliverance of health care, and for that reason call them: Medicine Men, the Mighty, the Meek, and the Mystic.

The first of these, Sir Hans Sloane, 1660-1753, was a mighty physician in his time but is known today much more as the founder of the British Museum, which together with such names as Sloane Street, Sloane Square, and Hans Place stand as monuments to this man. Like so many 17th and 18th century giants, he was a multifaceted scientist, naturalist, botanist, collector, investigator, physician to royalty and the poor alike—and a friend of buccaneers and pirates while in Jamaica where he experienced events which influenced his whole future. After receiving a doctor's education, loaded with botany while working with Magnol in France, he returned to London with a letter of introduction from Boyle, the great chemist, which he presented to Thomas Sydenham, the doyen of clinical medicine at that time. The letter stated, "Here is a ripe scholar, a good botanist and skilled anatomist." Sydenham appraised the 24-year-old doctor and said, "All this is mighty fine, but it won't do! Anatomy! Botany! Nonsense. Sir, I know an old woman in Covent Garden who understands botany better. As for anatomy, my butcher can dissect a joint full and well. No, young man, all this is stuff. You must go to the bedside. It is there alone you can learn disease."[2] Here is the first point for getting back to the basics: *Go back to the bedside.*

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* This is an abbreviated account of an illustrated after-dinner talk at the annual meeting of Kansas Chapter, American College of Surgeons, Topeka, November 12, 1977.

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So Hans Sloane went into practice, guided by his mentor, Sydenham; his future as a physician to the aristocracy seemed most promising. Furthermore, he was elected into the Royal Society at the age of 25, and as a Fellow in the Royal College of Physicians at age 27. However, amid those rosy prospects, he was seized by wanderlust prompted by an invitation to become the physician to the Duke of Albemarle, Governor of Jamaica.

Against the advice of Sydenham, Sloane made the trip across the Western Ocean to the British Islands of Jamaica, the gateway to the Americas. It appears that this 20-month sojourn significantly influenced the future of this brilliant man for it: (1) provided him with an unlimited opportunity to make detailed scientific observations of the flora and fauna of this new world; and (2) threw him into a close relationship with people of different walks of life, including pirates, natives and particularly African slaves, some of whom were destined for continued servitude in the new British colonies.[3] His detailed observations of these varied peoples and their customs and mores aroused in him a new empathy and he developed a new *social consciousness*—the second point in getting back to the basics.

The effect of this new conscience was notable in his practice upon his return to London. He became aware of the poor and established a personal practice of free care for the indigent in the mornings. He attempted to persuade the Royal College of Physicians, as well as the apothecaries of the day, to establish policies for the health welfare of the poor. He also took personal responsibility for the health of 400 orphans who lived in Christ Hospital, London. His practice was also expanding to include many aristocrats as patients, and for a brief period of time he assumed the role of what we now would call a clinical investigator. This was undertaken at the request of royalty, Princess Caroline, the Queen of James II. One of their children had narrowly escaped death from smallpox, and to avoid further such dangers, the Queen asked Hans Sloane to look into the possibility of "engrafting for the pox," as was rumored to have begun in Turkey. So, as seems the pattern of many researchers even in more recent times, he asked the Queen to release six convicts for this inoculation against smallpox. Sloane then, with the King's surgeon, arranged for the inoculation also of six foundlings (charity children). In the meantime, he had learned that in Boston, New England, among the 80 persons who had received the "engrafting" there were only two deaths. With this investigative data, the Queen then agreed to the inoculation of her other daughters, which it is said was successful.

Sloane's preeminence of that day is reflected in his elections to the presidency of both the Royal Society and the Royal College of Physicians. At the time of his death at the age of 93, he had influenced most of the notable personages of his day and had collected fantastic hoards of books, manuscripts and specimens of flora and fauna—many of which are described in detail*—that formed the basic original collection for the establishment of the famous British Museum. His monument in the Old Chelsea Parish Church is complemented by the streets and squares in Chelsea which bear his name.

The travels and observations of Sloane may have influenced Charles Darwin to make his own trip a century later. It is known that Darwin worked in the library room in the Chelsea Physick Garden which contains Sloane's account of his voyage to Jamaica. Young Darwin's "Voyage of the Beagle" was also a most important five-year experience which influenced not only his life, but the future tenets of both science and religion.[4] He had already half-heartedly studied for medicine at Edinburgh and for the clergy at Cambridge, but when the opportunity arose to assist Captain Fitzroy on a journey around the world, he succumbed to that attractive wanderlust; his father reluctantly consented to it only after an uncle's intervention on Charles' behalf. Darwin's objectivity in his observations and his personal convictions regarding natural selection of the species as determined by the environment led to the domination of his thoughts by pure physical phenomena.[5] As he grew older he became aware of this scientific enslavement in his living so that in his autobiography he said, "If I had my life to live over again, I would make it a rule to read some poetry and listen to some music at least every week . . . a loss of those tastes is a loss of happiness and is possibly injurious to the intellect and more probably to the moral character, by enfeebling the emotional part of our nature." So as point number three in getting back to the basics I suggest *Read some poetry and listen to some music every week,* so as not to enfeeble the emotional part of your nature.

One of Sloane's contemporaries was Sir Christopher Wren, the master architect of the City of London, and specifically of the magnificent St. Paul's Cathedral. Wren was highly skilled in astronomy and mathematics, but it is not well known that at one time in his life he showed great promise as a physiologist and researcher. His initiative and originality is evidenced by the fact that he was one of the first to experiment with intravenous

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* A two-volume set of Sloane's "Voyage to Jamaica" (1707 and 1725) is currently housed in the History of Medicine Library, University of Kansas Medical Center College of Health Sciences and Hospital, Kansas City, Kansas. A similar set of volumes was found in Darwin's room of the Chelsea Physick Garden, London.

infusions in dogs. With the use of a hollow pointed quill inserted into the femoral vein of dogs, he injected solutions such as wine, ale, and opiates! In view of these rather direct ministrations, perhaps it was a good thing that he was distracted from his research by a major exigency in 1666—the great fire of London which literally destroyed the beautiful old square-mile city. Commanded by the King, he could not refuse this great opportunity to employ his architectural abilities on such a grand scale. He redesigned the whole structure of the city, rebuilt the beautiful St. Paul's in much grander proportions, and with special craftsmen rebuilt 51 "Wren churches," which even now are shining gems of sheer beauty and design. Some are imposing in their position and location (St. Brides); others are tucked between modern buildings and are difficult to find but rewarding to the diligent pursuer. One has even been dismantled and reconstructed on the Westminster College campus in Fulton, Missouri, in honor of Sir Winston Churchill's famous "Blood, Sweat and Tears" speech. Over the grave of Sir Christopher in the crypt of St. Paul's is the following inscription: "If you seek his monument, look around you." This vignette illustrates our natural tendency to put off or procrastinate our promising research if any exigency (though much less dramatic than London's conflagration) arises. It is so easy to omit reading the articles or journals related to our patients' problems, or to delay that long-dreamed of investigation to some future time. This leads to point number four in getting back to the basics. *Don't let the distractions of the day keep you from your research.*

We may have something to learn from another of Sloane's 18th-century contemporaries, the Reverend John Wesley, the Founder of the Methodist Church, still considered a non-conformist church in England. Although John Wesley was not a physician in terms of the recognized authority (Royal College of Physicians) in England, he regarded good health in both physical and spiritual terms. He also recognized several problems in contemporary health care. The bona fide physicians of the day spent their time in the many coffee houses in the city receiving the apothecaries and the barber surgeons who described the symptoms of their patients. The physicians then prescribed a barrage of medications, herbs, roots, drugs, and purges—without having seen or examined the patients. Wesley deplored this practice and in his own way attempted to bring medicine to the people. Just as he departed from conventional in-church worship by bringing religion to the masses in the fields and factories, he was an itinerant preacher and "physician." But he recognized the danger of the polypharmacy of the time even more clearly, suggesting that such a multitude of therapeutic assaults might even make the patient more ill, and if the patient might by chance be improved, how would one know which of the medicaments was beneficial?

The tendency to multiple drug approach is returning today with the barrage of combinant antibiotics and chemotherapeutic programs. Furthermore, it is not unusual to find that our patients appear with lists and handbags full of accumulated medicines. This brings us to point number five in getting back to the basics—*Don't overtreat your patient.* Wesley believed so strongly in this that he published a best seller in his time, "The Primitive Physick," a large compilation of home remedies, all to be taken or tried singly; he covered himself by providing lists of four or five alternative remedies as well![6] The illnesses, if we can call them that, ranged from the control of hemorrhage after amputations to the treatment of common baldness, *e.g.,* "Rub the part twice a day until red with onions, and then honey . . . or electrify it." John Wesley did have his own electric machine and did use it; this was a common treatment practiced also by Benjamin Franklin and by Doctor Mesmer. It is interesting to note that an early traveling experience played an influential part also in the life of John Wesley—a trip to Georgia in the American Colonies, a territory of which Sloane was named a Protector.

Let's step now into the 20th century and recount another voyage which reveals some rudiments of basic medicine—a trip made to Sarawak in Borneo at a time of political unrest during the forming of the Federation of Malaysia in 1963.[7] I had gone there to help Dr. Norvin Schuman in his small Methodist 40-bed hospital in Kapit, a secluded inland village in the jungle. There, surprisingly, this mighty but meek physician was able to work in a synergistic fashion with the mystic medicine men of the area; there scientific medicine worked hand-in-hand with witchcraft. Scientific medicine there consisted of a primitive operating room, meager laboratory facilities for blood counts and urinalyses only, and a dedication to good clinical diagnosis and treatment. The people lived in the jungle with headhunting just a generation removed and still practiced sporadically on the Indonesian borders. Their houses were longhouses built on stilts, accommodating all the relatives of the family. Net bags of skulls still hung from the rafters in these houses. The only means of transportation consisted of longboats on the river which curls through the tropical jungle at temperatures and humidities over 100 F/ per cent. Our arrival in Kapit, after a 12-hr journey by launch, was one of intense interest to the small natives dressed sparsely and displaying their ubiquitous tattoos, just as Darwin had described a century earlier.

The diagnostic acumen and practical therapeutics which were so necessary were beautifully carried out by

the dedicated people of that hospital. Their relationships and attitudes toward the natives reflected tolerance and kindness; the tacit arrangement with the medicine men of the area practicing their witchcraft was one of remarkable cooperation. The Mission hospital regularly received patients who had been brought by an overnight jungle "ambulance"—a litter on a longboat with ill patients properly *referred* to the man of science by the medicine man. Patients so referred displayed a white cross marked on the skin, usually *where it hurt,* imprinted there by the witchdoctor. Several patients appeared with the white cross evident over the region of the enlarged and palpable spleen of typhoid fever. The medicine man had learned that Dr. Schuman could cure that malady in just a few days with his magical chloramphenicol. This arrangement functioned effectively, *doing the necessary* without the help of batteries of tests or diagnostic scans, but with the help of very practical mighty, meek, and mystical men of medicine.

In summary, and with tongue in cheek as we examine the lives of others, we can regain some of the pleasures and satisfactions of the practice of medicine by a posture inclined to get back to the basics. From these medicine men we reiterate what we have learned:

1. Sydenham—Go back to the bedside.
2. Sloane—Develop a social consciousness.
3. Darwin—Read some poetry and listen to some music every week so as not to enfeeble the emotional part of your nature.
4. Wren—Don't let the distractions of the day keep you from your research.
5. Wesley—Don't overtreat your patients.
6. Schuman—Do whatever is necessary.
7. Witchdoctor—Mark the spot where it hurts and refer your patient if you can't handle the condition.

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Augmentation Mammaplasty

(Continued from page 206)

never allowed to become an effective constricting force.

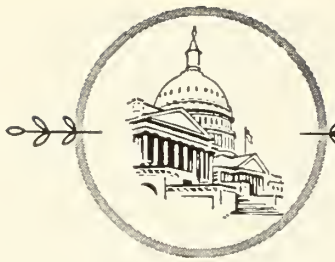
Finally, after the fact, how do we deal with an established capsular contracture? Surgical capsulotomy under direct vision appears to be the safest and most effective method. This involves an infra-mammary approach followed by initial incision of the capsule with an electric scalpel, which does not damage the prosthesis and allows its later reinsertion. Multiple relaxing incisions are made in the dome of the capsule as well as extensive enlargement of the cavity circumferentially at the base of the capsule. Some prefer what is known as an "external capsulotomy" which involves the application of a considerable amount of force to the breast in order to rupture the capsule. Theoretically, there would be some danger of rupturing the prosthesis as well, and there is no way to know immediately whether this has occurred. This procedure could possibly be utilized in dealing with slight firmness of the breast when the capsule is thin enough to break with less pressure.

Summary

Previously used methods for augmenting the breast purely for cosmetic reasons have been reviewed for their historical value. Present methods employ the retro-mammary insertion of a silicone membrane containing either silicone gel or saline. Complications of this type of surgery include a low incidence of hematoma, infection, numbness, ptosis, asymmetry, and skin erosion; far more frequent is the complication of fibrous capsular contracture, and this was discussed in detail with reference to its suspected causes, prevention, and treatment.

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Socio- ECONOMICS

How to Make Your Collection Calls Work

HOWARD E. WHEELER,* ***Topeka***

THE OBJECTIVE of all collection follow-up is to obtain payment in full of the amount owed. The effective use of the telephone can be an important part of the collection process—particularly when regular written follow-up procedures have failed to elicit payment from the debtor.

Every patient is entitled to receive from the doctor's office an itemized statement showing the balance he owes and how this balance was determined. This statement should indicate to the patient exactly what is expected of him in terms of payment of his account. If the account is to be paid in full within 30 days, the statement should indicate this. Because it is not always possible for the patient to pay his account in full in the stipulated time, the statement should also include an alternative for the patient; *i.e.,* it should state that the patient may contact the doctor's medical assistant to make arrangements for installment payment if he cannot pay in full within the specified period.

If the patient does not respond to this statement by making full payment or credit arrangements, several follow-up reminders should be sent to him. The follow-up communications may be statements, form letters, or dictated letters. Generally speaking, if the patient has the money to pay or wishes to make credit arrangements, he will respond within the first three or four months. Fortunately, about 90 per cent of all patients billed do respond within this length of time. However, those who do not respond probably will require motivation other than written communication to pay their accounts. At this point in the collection process, careful use of the telephone should be introduced.

Telephoning patients whose accounts have become delinquent should be entrusted only to experienced, well-trained medical assistants in the medical office. These employees should have a thorough knowledge of the doctor's credit policy; the fee structure on which all charges are based, and the details of all insurance and other third-party coverage. In addition, it is essential that employees who make collection telephone calls have a compassionate understanding of human nature.

One of the most important tools in persnading the patient whose account has become delinquent to pay in full is the collection telephone call. However, if not handled properly, the telephone call to obtain collection can be useless. Practice has shown that the effective collection call follows a definite format. Here's a study of the anatomy of a successful telephone call.

Telephoning the patient with a delinquent account is not merely a matter of making a contact and demanding payment. Statements and letters have already done that. Lack of response to these preliminary collection communications indicates that the patient is either unwilling or unable to comply with a demand for payment. However, the telephone is an instrument of two-way communication, and proper use of the telephone can reveal why previous communications with the patient have gone unanswered.

The collection call should follow a specific sequence, which is essential to the success of the call. The employee who makes the collection call should be thoroughly versed in the sequence of steps in the effective collection call before the call is ever made.

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The first step in the sequence is to positively identify the debtor. The person making the collection call must be certain that he is actually talking to the debtor, not to another member of his family who has a similar name, for example. As soon as the debtor's identity has been established, the collector must identify himself—by name. He should not say, "This is ABC Clinic calling." Only a person can make a telephone call. At this point in the collection call, it is best for the collector to emphasize his name, rather than that of the institution, because the debtor can relate more easily, and with less fear, to another person than he can to an impersonal clinic or professional association. In fact, it may not even be necessary to identify the institution until later in the telephone conversation. To obtain maximum results from the call, it is preferable that the collector try to maintain a "one-to-one" relationship.

Once the collector has determined that he is talking to the debtor and has identified himself, he should ask the debtor, "Do you have a few moments to talk with me?" Asking this question will pay many dividends. In addition to being an act of courtesy, it will tend to establish confidence and rapport between the collector and the debtor.

If the debtor indicates that he does have time to talk with the collector, the collector should immediately state the exact purpose of the call. He may say something like, "I am concerned about your past-due account with our doctor's office. It amounts to $160, and it is now five months overdue. The doctor's accountant has asked me to talk with you about this because he feels that you are probably just as concerned about it as we are. Perhaps I can be of some help. Will you send us a check for the full amount today?"

With these brief remarks, the collector has stated the facts of the account; has indicated that he thinks the debtor is concerned about settling his account; has offered him assistance in paying the account, and has asked for the balance in full.

At this point in the call, the collector should stop talking. He should allow the debtor to talk. This is known as the "psychological pause." During the next few seconds or minutes, the collector will learn many things. To begin with, he should be able to determine the debtor's attitude toward him and the doctor's office. If the debtor has a complaint, he will make it now. He will give his reasons for not having paid, and will probably attempt to justify his position. It is likely that he has been expecting a telephone call from the doctor's office—probably long before this time. He is likely to feel guilty, which may cause him to experience one of several reactions, including anger, hostility, or even fear.

It is important at this juncture that the collector overcome the temptation to be irritated with the debtor. He should not belittle the debtor's excuses, justifications, or problems. By all means, the collector should not preach to the debtor. The debtor knows he is wrong, and pressing him to admit it will accomplish nothing except to put him on the defensive.

The collector will be more successful if he waits until the debtor has finished talking and then takes over in a positive and constructive way. He should make every attempt to control the rest of the conversation without being overbearing. The collector should weigh the objections the debtor has raised and counter them with facts, logic, or intelligent questions. For example, if the debtor insists that insurance should have paid the bill, the collector should obtain specific information from him so that this possibility can be verified. After all, it is possible that the doctor's office has made a mistake by failing to obtain the necessary forms and forward them to the insurance company.

Throughout the collection call, the collector should use the "you" approach, rather than emphasizing such words as "we" or "I." The debtor is much more interested in his own welfare, his credit rating, his problems, his reputation, etc., than he is in the immediate problems of the doctor's business office.

As the call progresses, the collector should obtain as much additional information from the debtor as possible. He should find out where he lives, where he works, what his supervisor's name is, whether his wife works, if he has other credit problems, etc. The collector should also attempt to determine whether the debtor's problem has a time limit. In other words, the debtor's situation may improve within a reasonable length of time. If so, it is up to the collector to find this out.

If the collector is satisfied that the debtor's present circumstances are indeed difficult, but that the situation is likely to improve relatively soon, it may be wise to work along with the debtor until the situation actually does improve. On the other hand, if the collector feels that debtor is stalling, he must be firm and insist that the debtor face the problem at the present time. He must sell the debtor on the benefits of paying off the account in full as soon as possible. If the debtor expresses doubt about his ability to pay the balance at the present time, the collector must instill in the debtor confidence in his ability to pay. The collector may point out to the debtor that he has handled many similar situations and has always worked out satisfactory solutions when he has had the full cooperation of the debtor.

The collector should never argue with the debtor. Absolutely nothing will be accomplished by proving the debtor wrong. Instead, the collector should listen for "closing clues" throughout the conversation. These clues, indications of what is likely to motivate the debtor,

may be used in the final stages of the conversation to secure a definite promise to pay. For example, the debtor may state emphatically that his family (or his children) come first. This statement is a clue to what motivates him. The collector may then remind the debtor that by paying this account in full, he can ensure that full medical treatment will always be available for his family. This reminder is likely to be successful in effecting collection because concern for his family is what prompted the debtor to make the remark.

As another example, if the debtor expresses concern that he may be called at his place of employment, the collector may interpret this concern as a strong closing clue. He should assure the debtor that he will not consider calling him at work as long as the debtor cooperates. Again, if the debtor asks whether he has to pay the full amount, the collector may assume that the debtor really wants to do the right thing, but that he will need firm direction from the collector.

The collector should constantly bear in mind that the primary purpose of a collection telephone call is to obtain a valid promise from the debtor. The collector cannot get the money instantly, so he must insist upon a reasonable promise to pay the full balance or to make arrangements to pay in installments. Should the collector encounter a problem case that he simply cannot handle effectively, he should not hesitate to turn it over to someone else in the business office. Many times, a new voice and a fresh approach to the same debtor will produce the desired results.

To return to the collection call, once the collector has obtained from the debtor a reasonable promise to pay, he has reached perhaps the most important part of the call. At this point, the collector must be absolutely sure that the debtor fully understands exactly what is expected of him. If he does not understand this, it will be necessary for the collector to make a subsequent call to the debtor.

The debtor and the collector should agree upon the following:

1. The exact size of the expected payment. The collector should never be satisfied with statements like, "I'll send in whatever I can spare," or "I'll do the best I can." The collector must make sure that the debtor agrees to pay a specific amount.

2. The exact time when the payment will be made. The collector should not settle for declarations that payment will be made "at the end of the month" or "sometime in the next two weeks." The collector is entitled to know the exact day of the week, the exact date in the month, and even what time of day that payment will be made. If the collector settles for anything less specific, he has a poor promise.

3. The exact mode of payment. The collector should find out whether the payment will be made by cash, check, money order, etc. He should also know whether it will be mailed or brought into the office.

4. Exactly who is going to make the payment. The collector should find out whether the debtor himself will make the payment, whether his wife will, etc.

After the collector has secured agreement on all four of these points, it is extremely important that he repeat the arrangements to the debtor. If there still appears to be some question in the debtor's mind, the collector is wise to suggest to the debtor that he get pencil and paper and write down the arrangements.

Finally, the collector should make absolutely sure that the debtor has the correct mailing address of the doctor's office or clinic. If he feels it is necessary, the collector should offer to send the debtor a stamped, self-addressed envelope in which to make the payment. This envelope should be marked for the personal attention of the collector. This little detail can, surprisingly enough, produce a marked increase in collections.

It may seem that the telephone sequence described here presumes that the debtor is somewhat childish. The point is that the collector sets out to make an effective collection. If repeating details two or three times will make the call more effective, then the repetition is certainly worth while. After all, advertising experts have discovered that constant repetition is effective in accomplishing their objectives.

The analogy between advertising and collection may be carried even further. A collector is very often in direct competition with other telephone collectors—for the limited amount of money the debtor possesses to pay his obligations. In fact, the debtor may have become delinquent in his payments in the first place because he does not have enough money to go around.

Thus, if the collector is to be successful, he must make a positive impression on the debtor during the first collection call. He must secure the debtor's confidence; demonstrate understanding of the debtor's problems; sell the debtor on the benefits of paying his account in full, and make certain that specific arrangements are made with the debtor—arrangements which the debtor clearly understands. In addition, the collector should make it as easy as possible for the debtor to keep his agreement to pay. Throughout the call, the collector should treat the debtor fairly but firmly.

Finally, if the collector can bring the debtor to feel that he has someone to turn to in the doctor's business office if he has problems in the future, then he has accomplished his goal. He has used the telephone effectively for collection.

The President's Message



At the sixth annual AMA Leadership Conference held in Chicago, January 26-29, 1978, the results of an AMA-commissioned survey of Doctor and Patient Attitudes were studied.

Doctors view themselves as technicians and as different from other professionals. The patient's (consumer's) problems are perceived by the physician from the standpoint of a technician, and not from the patient's viewpoint. Could this be a result of the problem oriented record? The doctor talks to the patient/consumer as a technician, not as a concerned professional.

The surveyed doctor is also concerned with the paperwork of a variety of government programs, but expresses little concern with where such programs are headed.

These opinions are not shared by everyone in the medical profession. They do, however, give a very interesting picture of a turnabout in our view of the profession, a view that has something to do with our present and future difficulties with health care in the United States of America.

The patient/consumer survey revealed that physicians still have the public's trust and confidence. The consumer is more concerned with the personal relationship he establishes with his doctor than he is with the doctor's technical knowledge and skills (recent malpractice suit claims to the contrary).

Governor Brown of California, in a recent talk to the National Health Forum sponsored by the National Health Council in San Francisco, suggested that one way of bringing "human values" to the health care system would be for physicians to "stop the strong worship of technology. This is not just true of doctors, but politicians as well."

How can we explain this apparent paradox? The patient seemingly wants to preserve the old family doctor image. The doctor, however, sees the patient as a problem to be solved, and not as a person, and has more interest in the problem than in the person.

The ability to do more for the patient with the rapid and massive growth of technology; admission committees more interested in grade point averages than personalities; medical school curriculum that stresses specialties over family practice; medicine as a business, not a profession; humanism as the dominant philosophy —all these and more have been used to point the finger at THE cause; but doesn't it really lie with you and me?

Is this the time to restudy the objectives, ethics, philosophy, and codes of the medical profession? Is there room in a humanistic society for a code that was developed on humanitarian principles? While there are many problems that lie beyond our ability to solve or influence, these questions are ours—yours and mine—to solve.

Am I a technician or am I a doctor (with all the idealism that profession used to imply and probably still does in many cases)? Do I really practice what I believe? Is there room for both? Someone has said that we act as we believe and think! It may be that Pogo is right, "We have met the enemy and he is us."

Fraternally yours,

Warren E. Thayer, M.D.

President



Solution

Periodically the passing scene spawns certain word combinations which are taken up in the history of the time and provide for posterity a glimpse of the spirit of that day—Fifty-Four Forty or Fight—Tippecanoe and Tyler Too—Remember the Maine—The Great Society—Farrah Fawcett-Majors. Of late, a new term has appeared which may well take its place among those immortal phrases that have brightened the lexicons of the past: Cost Containment. This term has been devised to describe a policy roughly equivalent to that established by the irate householder who emerges from the monthly bill-paying session shouting, "What the bleep's going on around here?" Its current popularity stems from the belief that health care has been taking too big a bite out of the economic pie.

Several suggestions have been made for the implementation of this policy of containment with, as yet, no remarkable evidence that the populace is unanimous in rallying to the cause though it continues to lament the status. The administration, misreading somewhat the political tea leaves, tried putting a lid on hospital costs to no avail. The National Commission on the Cost of Medical Care has, however, displayed admirable courage by indicating that the prime effort must come from the conscious determination of those two principals in the process, the patient and the physician, to reduce expenditures. Although the medicine is appropriate and has been prescribed in a forthright manner, skeptics who have observed the habits of these two characters over the years may doubt that the prescription ever gets filled. Still, it is the only thing that will finally work, and that is why we have been working on a plan to accomplish this end.

From time to time in the past, we have offered a number of solutions for the problems of the world. The astute analysis and practical wisdom of these offerings is witnessed by the fact that the world has not seen fit to accept them and consequently, as you will note, is still beset by those problems. Undaunted, we once more present a simple and effective method of achieving cost containment: eliminate the third party payer. To some this will seem to be of a sort with the advice of Dick the Butcher: "First, let's kill all the lawyers," an idea not without merit but for another time. No, we're not advocating anything so drastic for these third parties but the idea does address the basic cause of our present griefs and, judging by the sounds being emitted by all three of the parties, the suggestion should be generally welcomed.

First, we must go back to the time before the third party entered the scene. Then it was the physician and the patient fighting the battle for health side by side. This was the Golden Age of the Physician-Patient Relationship—at least we guess it was. Everyone keeps talking about how great things used to be but nowadays, next to the high cost of medical care in general and the physicians' astronomical incomes in particular, what do we hear more about than the loss of that sacred relationship? Oh, the change came easily enough and with good intent. In those days, when Joe's rupture got to bothering him so he couldn't work, Doc repaired it so he could work. But Joe couldn't pay much of anything, him not working regular and the missus being in a family way again and all. So Doc took what he could get, even if it was just a sincere promise to call him when the missus was laboring again—and the p-p relationship was intact. Since Doc wasn't starving, it was more or less understood that those who did pay him were somehow taking up the slack for Joe.

Then that young insurance salesman came into Joe's boss's office to leave a girlie calendar and stayed long enough to sell the boss on the idea of buying a policy that would take care of Joe's medical expenses as an inducement to Joe to put in most of a day's work. Seemed all right to Joe—didn't seem to cost him anything and while he figured Doc would take care of the missus when her time came anyway, maybe he wouldn't act so grumpy about it if he got paid something. After Doc

got over his shock, he agreed the idea was of some merit beside its novelty. He had always prided himself on taking care of Joe and all the gang (up to a point), feeling that warmth that comes with serving humanity without thought of remuneration (up to a point). He decided some payment wouldn't nullify the feeling altogether and anyway what he got wasn't up to his regular fee. The insurance man was happy with the sale but had a few bad moments when Doc and some of his colleagues who figured the insurance man was making a killing (pardon the expression) set up a so-called nonprofit system with the advantage that they determined the benefit themselves. It became apparent, however, that there was enough to go around even as more people got into the act, and the insurance company was able to hold its own against the Blues and still have enough left over at the end of the year to buy a baseball team.

No one actually said, "Let's blow that corny patient-physician relationship." It just sort of got stretched a little to accommodate this third party and then seemed to keep spreading. As it happened, Joe and Doc were getting so tied up in their new roles that their relationship with each other just seemed secondary. What really clobbered it was the government getting into the act. Oh, the local officials had been hassling with Doc off and on about taking care of the folks at the county home, but no one thought of this as insurance. But the government boys kept finding more folks who ought to be looked after and explained patiently to Doc how to do it—since, as they said, they were paying him. They brought their tested and proven snafus which were much more sophisticated than those the county used and managed to separate Doc and Joe more, as Damon Runyon used to say, than somewhat. And somehow, the old relationship wasn't helped much by the weekly news releases about Doc's penchant for abuse of the system if not downright fraud.

Well, as long as everything was being paid for anyway, Joe came to see his health care as a right rather than a privilege (what it was, Doc was really keeping him in a sort of slavery) and came to demand daily miracles regardless of the cost. Technologic wonders that would have evolved more slowly in a less enlightened, pay-as-you-go age burgeoned. Increasingly sophisticated hardware was made obselete each year as the new models came in (in the best Detroit manner). Joe had never been worth so much in his life considering the cost of keeping him patched up through the grace of those magic words, "It's covered by insurance."

Doc was puzzled. Here Joe was getting the best of care—all the most expensive diagnostics and therapeutics

(Continued on page 231)

Vox Dox

Vox Dox Editor:

In his book, "The Horse and Buggy Doctor," published in 1938, "Pa" Hertzler states:

> To socialize medicine is to remove the human touch. . . . In fact, the public demands nothing except the privilege of patronizing quacks and cults. Higher medical standards are entirely the work of doctors themselves—conscious of their own limitations.
>
> Let it be repeated here that the advance in medical requirements has been autonomous action by the medical profession, and not in response to public demand.

That statement was made 40 years ago. What is the situation today?

The Kansas Legislature is about to legalize the sale of laetrile (by popular public demand) to cancer victims. The bill is being slightly amended to prohibit the selling of laetrile without a prescription from a physician and further amended to delete from the record keeping requirements the provision that would have required physicians to record the name and address of the patient receiving laetrile and the reason for dispensing laetrile to such a person.

Legalizing laetrile by popular public demand is a terrible tragedy in itself. For even one physician to lower his medical standards to sign a prescription for such a drug compounds the tragedy to double its enormity. If no physicians can be found who can be enticed into signing such a prescription, the legislature might take it upon itself to make a law forcing the physicians to sign such a prescription upon the demand of the patient. This would triple the tragedy. In fact, it would nullify the present law requiring every physician to complete so many hours of continuing education or lose his license to practice medicine, and in effect would follow the trend the legislature is now taking—that "you will now practice medicine according to the popular public demand and not by any scientific facts you may gather in courses of continuing education."

What a sham and farce this would be!

A. S. Reece, M.D.
237 W. Main
Gardner, KS 66030

Editor's Note: The Kansas Legislature passed the bill legalizing the manufacture and sale of laetrile on April 26, 1978.



Personalities —IN KANSAS MEDICINE

David G. Laury, Ottawa, immediate past president of the Medical Society of the Pan-American Doctors' Club, presided at their recent annual meeting, where he was elected to the board of directors.

The Kansas State Society in Washington, D. C., has named **John B. Jarrott**, Hutchinson, as recipient of their 1978 Outstanding Kansan Award.

KMS members who recently testified before legislative committee hearings on the legalization of laetrile included **Jimmie A. Gleason**, Topeka; **David E. Street** and **Harry E. Hynes**, Wichita.

Robert F. Sinclair, Manhattan, director of Lafene Student Health Center at Kansas State University, has announced establishment of an alcoholism prevention campaign funded with a grant from the Kansas Department of Social and Rehabilitation Services, Alcohol and Drug Abuse Section.

James W. Wilson, state director of health, recently testified before the Public Health and Welfare Committee of the Kansas House of Representatives in behalf of a modernization of the Kansas Nurse Practice Act.

Katherine Pennington, Wichita, recently authored an article on child care procedures for the Wichita *Eagle*.

Daniel J. Stechschulte, program director, and **Dennis Finley**, Kansas Arthritis Foundation president, have announced receipt of $20,000 funding in grant awards for 1977-78 by the Arthritis Clinical Research Center at KUMC.

KMS members who have recently hosted medical students under the auspices of the KUMC outreach program include **Edwin D. Rathbun** and **Richard L. Nevins**, Liberal; **Dale W. Anderson** and **James L. Barber**, Augusta; **F. G. Freeman**, Pratt; **Martin B. Klenda**, Beloit; and **Earl D. Merkel**, Russell.

A recent workshop on "Allergies in Children" presented by Newman Memorial County Hospital in Council Grove, featured **Karl K. Kavel**, Topeka, as guest faculty.

Earl B. Gehrt, Chanute, Neosho Memorial Hospital chief of staff, recently explained emergency room policies for readers of the Chanute *Tribune*.

Law enforcement personnel from the Pittsburgh area heard **William G. Eckert**, Wichita, discuss forensic medicine as it relates to legal evidence at a recent seminar at Pittsburg State University.

George Dyck and **E. G. Burdzik**, Topeka, have been cooperating with officials in Memphis, Tennessee, in an attempt to verify the identity of a young girl believed to have been a recent patient at Topeka State Hospital.

R. R. Brummett has closed his practice in Neodesha and moved to Salina, where he has joined a group practice.

Barbara Lukert, M.D., recently became the first woman president of the Wyandotte County Medical Society.

William Reals has been named Assistant Dean for Research, Development and Planning at The University of Kansas School of Medicine-Wichita. He will coordinate faculty research activities and serve as chairperson for the UKSM-W research committee.

PHYSICIANS IN HISTORY

About 3:00 A.M. on Wednesday, May 21, 1856, Florida's Colonel Titus, leading a troop of about 200 horsemen, secured the crown of Mount Oread. Between morning's darkness and the early light, the remaining forces of the Upper Division—which had been encamped 12 miles north at Lecompton—moved into position. Most were on horseback. The leaders accentuated their commands with proslavery aphorisms punctuated with vile profanity. General Dave Atchison—former Missouri senator, President of the Senate, and Vice President of the United States—led his troop of Platte County Rifles drawing two artillery pieces; Captain Dunn led the Kickapoo Rangers; General B. F. Stringfellow along with his brother, J. B. Stringfellow, M.D., led another troop, followed by the Doniphan Tigers. On the slope of Mount Oread, facing the settlement of Wakarusa (now called Lawrence), some of the troops occupied the residence of Charles Robinson, M.D.; others laid their field pieces aimed at the town below.

Alabama's Colonel Buford, leading the Lower Division—a troop of volunteers he raised and supported from the sale of a parcel of slaves—had encamped at Franklin four miles southeast, and now rode into position.

The besieging Army was now assembled and in position. They raised above their heads a blood-red banner emblazoned with "Southern Rights" on one side and "South Carolina" on the other.

J. B. Donaldson, United States Marshal of the Territory, a federal appointee under President Pierce who was charged with the responsibility of upholding the government of the territory, stood in charge of this *posse comitatus* committed to the rectification of a grave social evil.

The Free-States had elected Dr. Charles Robinson Governor of their party. He had come to Kansas as an agent of the Massachusetts Emigrant Aid Society, an organization heavily financed by Amos Lawrence and conceived by Eli Thayer, founder of Oread Institute, a Massachusetts women's College. The society endeavored to quiet antislavery sentiment in the newly opened Kansas Territory.

Free-State citizens had interceded and released prisoners where it seemed to them that Sheriff Jones had arrested some of their members under arbitrary and capricious circumstances. Provocation of antagonism seemed a part of the sheriff's lifestyle. Besides, as a resident of Missouri and a former Postmaster in Westport, they seriously questioned the legality of his position as Sheriff of Douglas County.

Jones had been shot in the back by an unknown assailant while sleeping in a tent at Lawrence. The Free-State leaders publicly denounced this wanton attack on Jones, offered a reward for the apprehension of the guilty one, and nursed Jones back to health in the Free-State Hotel. This incident at least gave Jones grounds for revenge.

Rumor spread wide in the Territory that the blood-thirsty abolitionists had assassinated Jones. The cry of the opposition and a prior siege had led Lawrence residents to erect fortifications for their personal protection. They had built four or five circular earthworks a hundred feet in diameter and seven feet tall, interconnected by trenches and fortified with rifle pits. The Free-State Hotel housed two newspapers, the *Herald of Freedom* and the *Kansas Free-State,* that published views in opposition to those in the *Squatter Sovereign* and comparable proslavery publications. Rumor now depicted the Free-State Hotel as a fortress with rifle slots and cannon holes which posed a military threat to the integrity of the proslavery government.

W. P. Fain, Deputy U. S. Marshal, and his assistants, now rode into Lawrence in the forenoon for the arrests and for the conscription of a citizens' posse to assist in the arrests. Fain expected bold military resistance; indeed he met courteous, cooperative compliance. He dined without expense at the Free-State Hotel and then returned to the summit of Mount Oread with his prisoners. Marshal Donaldson's mission completed, he now relinquished his *posse comitatus* to Jones for the next part of the mission.

The border patrol inspected everything flowing into Kansas, confiscated what they considered a threat to their cause, and intimidated potentially unsympathetic settlers into a prompt departure. Dr. Robinson had arranged through the Massachusetts Emigrant Aid Society for the shipment of Sharp's rifles to the Free-Staters, and to fox the patrol they were shipped in boxes marked *Bibles.* Since Henry Ward Beecher—brother of Harriet Beecher Stowe who wrote "Uncle Tom's Cabin"—raised money from his congregations for the rifles, they became known as "Beecher's Bibles."

Jones and 20 armed men rode into Lawrence where they confronted General Pomeroy in front of the Free-State Hotel. "You have five minutes to give up your arms or I will bombard the town," Jones declared in his usual gruff territorial language.

Pomeroy turned over a brass howitzer and a few spare arms to Sheriff Jones with the reply, "As you see, the remaining rifles belong to the citizens of Lawrence. I have no authority to deprive citizens of their personal property, particularly property essential for their subsistence and survival."

Jones retorted, "The district court has declared the Free-State Hotel and the newspapers therein to be nuisances subject to removal, and the court has placed the execution of these indictments in my hands. You will vacate the hotel immediately in preparation for the execution of these orders." Having voiced the stipulations. Sheriff Jones and his forces proceeded to destroy the two printing presses, demolish the Free-State Hotel, burn the home of Dr. Charles Robinson, and loot the town.

Tyranny won a fleeting local victory that fateful day, but strategy ultimately won by enlisting the more lasting and sympathetic support of the nation. Ironically, two casualties occurred, both among the invaders. One died of a shoulder wound after his rifle accidentally fired; the other died from a head injury that occurred when the South Carolina banner swept a loose brick from a building.

Opinion was not so sharply divided as the militant contest might at first suggest. The large plantations repeatedly raising only one or two crops supported slavery, but only as an unskilled economic tool. The highly technical and innovative economy would not support such bondage. These required the franchises of freedom that support individuality, responsibility, and ambition. Most citizens in proslavery states could not afford the large plantations nor the parcel of slaves sufficient to operate them. Some owned only a few or none, and maintained sympathies for emancipation; some sought a route to an orderly transition. Congress had banned the importation of slaves and heard the proposal that all children born after a cut-off date would be born free. The militant, tyrannical government operated under an unendorsed authority, and the proslavery forces supported the "Border Ruffians" who required no rationale for violence.

Among the Free-Staters, opinion likewise differed. Some felt the franchises of citizenship, particularly those set forth in the Constitution, belonged to everyone, slaves included. This after all was the reason for the American Revolution. "Free the slaves, but keep them out of Kansas" became the theme of others. Some even saw a middle ground and favored a compromise.

Unfortunatedly, the militant polarization of the two factions precluded free exchange of opinion.

Times, faces, and views have changed. Old issues, once resolved, have opened the way for new issues and solutions for these issues. Negotiations between minds that represent the voices of the people, negotiations that take place at the bargaining table in government—this at the moment seems to enjoy favor over violence. Both exact a price, but only one exacts a voluntary contribution. PAC monies support the voluntary process of representative government.—*J.R.C.*

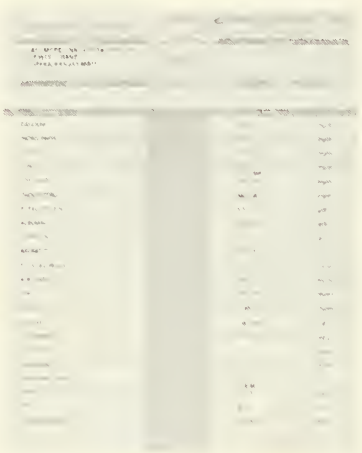
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Auxiliary News

This final letter to you should enumerate the accomplishments of your Auxiliary this year. I will try to include most of them in my convention report that will appear in the June issue of THE JOURNAL.

I find myself looking ahead at a time when reflection is appropriate. A nagging thought that has been with me all year is that the state and county auxiliaries can and should do more to serve their parent organizations. The major drawback is that they do not always know what needs to be done. The state and county medical societies can best evaluate areas that need Auxiliary talents.

For the first time in history of the AMA and its Auxiliary, the Association sought the help of the Auxiliary to conduct a nationwide project for the profession—the Immunization Awareness Campaign. According to the latest reports, it was a gigantic success. Not only did the campaign receive desired results in the immunization of children, but there were some spin-off benefits to the profession. The visibility of the medical family actively involved for the public welfare on a volunteer basis was some of the best public relations ever received. As one little boy said to the hopscotch painting doctors' wives, "Where have you been? We've been waiting for you." He was referring to the TV announcements.

While many physicians and their spouses serve in various organizations and on numerous policy-making boards, the profession is rarely credited with their contributions. Only when the professional organization functions does the public become aware of the personal commitment beyond professional responsibility. The old adage, "Actions speak louder than words" is still relevant. Somewhere on our list of priorities we should include professional efforts to serve on a volunteer basis.

An example of a joint Society and Auxiliary effort is developing in Johnson County. The Johnson County Medical Society and Auxiliary, in cooperation with the Heart Association and local business organizations, have a goal to train in CPR every county adult citizen and high school student. The Society and Auxiliary have each purchased a mannequin while business groups are funding others. Several doctors' wives who are nurses are taking instructor training (26 hours) and others are taking the 8-hour course for certification. Public instruction will be held at various locations in the county and the student courses will be given in high schools. Local media are aiding by supplying publicity for the project.

Perhaps not every county needs a CPR course, but every county has an unmet need.

The Auxiliary Learning Center on Body Pollution was exhibited at the national meeting of American Alliance for Health, Physical Education and Recreation, April 7, in Kansas City, Missouri. The AAHPER is an organization of teachers who share these areas of instruction at elementary, secondary, and college levels. Mrs. Charles Casteel, health education chairman, and I answered their inquiries about the games on tobacco, alcohol, nutrition, and drugs. The colorful unit was originally developed by the Wyandotte County Medical Auxiliary. It has now been duplicated by the state Auxiliary for use by 4th, 5th, and 6th grade students over the state.

A parting thanks to you for pausing to read this page. My offerings this year have been a labor of love but labor nonetheless. My typewriter has been offsetting its capitals and otherwise showing signs of fatigue, so rest is in order for us both. Please know that the Auxiliary is in capable hands as Jean Crouch takes command.

Sincerely,
Jackie Burnett
Immediate Past President
Kansas Medical Society Auxiliary



Book REVIEWS

THE FREEDOM TO DIE, by O. Ruth Russell, Ph.D., Human Sciences Press, New York, Rev. 1977. 352 pages. $14.95.

Death! The experience comes to all, and sooner or later it touches the lives of most, leaving a memory so vivid and laden with emotions there seems little doubt about what it really is, but try to define it with words that suit all occasions, and problems arise. Deprive anyone of a year, a month, a day or even an hour of their natural life, and the law says the depriver is a culprit, criminally liable. The natural span may be hard to determine, but the intent incriminates, and whatever the physician does or does not do can be viewed in an incriminating light. Give a patient a shot of morphine to relieve pain and another may view the drug as a respiratory depressant that shortened his natural life by an hour, a day, or even more.

When in former years an organ failed, everyone expected death. Now that modern medicine has dialysis machines to replace failed kidneys, respirators that mechanically ventilate silent lungs, and cardiac massage that pumps blood through a motionless heart, everyone expects to live, sometimes beyond the measure that is truly theirs. These procedures appear miraculous when a person who would have been dead a few years ago can quickly return to his family, friends, and employment again. But add to these measures artificial sustenance, unrelenting specialized care, a physiological state that cannot improve and does not deteriorate, and what remains? What choices are available—to whom, and when?

Some physicians deal with death repeatedly; others seldom see or feel its impact. It doesn't take much knowledge of physiology to realize malnutrition induces death slowly, starvation more quickly, lack of water faster, and suffocation in minutes. The physician who repeatedly treats the dying patient comes sooner or later to realize in the hopeless case his treatment sometimes only changes suffocation into starvation, and then malnutrition or even stabilization until long-term deterioration or an insolvable complication completely thwarts further treatment. As bones begin to show through the flesh, as smelly bed sores begin to appear, and as the cost begins to erode savings, home, the children's education and the nest egg for retirement, then the dignity in death becomes meaningful and realistic.

If a person has the right to die as a hero, an American, a Christian, or without the administration of blood, does the person who desires this choice have a right to die in accordance with an image held in life and before the process of dying erodes away that image?

In another era, many marveled at the pen of Emile Zola when he wrote: "No greater love hath he, than one who would lay down his life for a friend." In this generation of organ transplants we have come full circle to the point where it can now be said "what better love can a person have for the disadvantaged than he who would in his uselessness donate his viable organs to the needy." But how can this be done and who will do it? Time is important for the successful harvest of useful organs; yet, in death, the ownership of the remains so often passes to the nearest of kin or the one willing to assume financial responsibility for disposition of the remains. The choice, formerly of little concern, passes from the individual to others unless a valid choice can be made before death.

Euthanasia, while it means an easy death, is a bad term. Dr. O. Ruth Russell admits this, and she recognizes the need for a better expression. Her book, written in a very readable style, discusses this—the freedom to die. I would recommend its reading. The book, first published in 1975, has added a chapter covering interim events and their impact. Since this appears after the appendix (and I am not sure this is the appropriate place for it) some casual readers might miss this chapter.

To friends and acquaintances of the late Dr. William P. Williamson, formerly Professor of Neurosurgery at the University of Kansas School of Medicine, it may be of interest to note Dr. Russell mentioned with feeling, understanding and concurrence his views on death and dying.

Dr. O. Ruth Russell wrote and then revised this excellent monograph at a period in life when most are content to rest on their laurels. She deserves a hearty commendation for the example she has set for the continued use of valuable talents.—*J.R.C.*

Month in Washington

The Federal Trade Commission has charged that the nation's Blue Shield plans are dominated by physicians —"an arrangement that may reduce competition and raise prices artificially."

FTC Chairman Michael Pertschuk told the Interstate and Foreign Commerce Committee's subcommittee on Oversight and Investigations that "—it is difficult to see how the public interest can be served by such an apparent conflict of interest."

He said that an ongoing FTC investigation has found that "most" of the 72 Blue Shield plans are controlled by local medical societies, other physicians' groups, or "self-perpetuating physicians boards" set up to run the plans.

Subcommittee member Albert Gore, Jr., (D-Tenn.) said that many members of Blue Shield Boards of Directors "also serve on the boards of banks and lending institutions holding Blue Shield funds. These persons also have a direct interest in seeing that these financial institutions make a profit. I believe this practice is unconscionable and is an abuse of the health plans obligations to their customers," Gore said.

"It also poses an even more serious potential for abuse of the federal Medicare and Medicaid programs. Funding for these programs is distributed through Blue Shield organizations under contract with the federal government," the Tennessee representative said.

The FTC Chairman said his agency was limited in the actions it could take against insurance firms and non-profit institutions. The FTC lobbied hard in the last session of Congress for expanded authority, but the proposal to give the agency powers against non-profit institutions did not clear the House Commerce Committee.

Mr. Pertschuk acknowledged the need for physician input into the management of Blue Shield plans but said, "there is the danger that even a small bloc of physicians could dominate a larger group of lay people. And in light of the fact that commercial health insurers are able to provide medical coverage without physician directors, it is not obvious that any physician participation in decision-making functions—as opposed to advisory functions—is necessary at all."

The FTC Chairman again made a strong pitch for expanded agency authority, saying that the FTC Act "should be modified to give the Commission jurisdiction over all business entities, regardless of whether or not they are organized for profit."

The Health, Education and Welfare Department will release a list of all Medicare payments to all physicians despite warnings from both Congressmen and the AMA that a simple listing of dollar amounts paid to physicians, with no indication of the number of patients treated and the services provided for those payments, is essentially meaningless.

The list, to be available for public inspection, containing the names of some 300,000 physicians who provided services to Medicare-eligible patients during 1977 and reported to be some 5 ft. thick and costing "perhaps as much as $1 million"—will tell nothing about the physicians named, the kinds of care they provide, their actual earnings, or the patients they serve.

The list will not indicate, for example, that across the country the doctors actually received, in Medicare reimbursement, only about 58 per cent of the amount of covered charges actually billed for providing medical services to persons whose care is paid for by Medicare.

In a letter of protest to HEW Secretary Joseph Califano, AMA Executive Vice President, James H. Sammons, pointed out that "There is no cost-benefit ratio in what HEW is doing. The interests of neither the public nor the profession are served by this type of reporting."

Dr. Sammons spells out in the letter many of the flaws and down-right inaccuracies of a list compiled in such a fashion. For example, the list will not make clear that in many instances the payment for covered services is made directly to patients who are then supposed to pay the physicians, but often do not. The physicians are credited by the HEW list with having received all moneys paid for services which they provided, but in countless instances they may have received only a part of it, or none of it.

Dr. Sammons said "—the list will not explain that certain specialists such as cardiologists, internists, urologists, nephrologists, surgeons, ophthalmologists, or orthopedists will naturally have large numbers of elderly or chronically ill among their patients, all covered by Medicare. Nor will it indicate that physicians located in Florida, Arizona, California, and certain other states are far more likely to have a Medicare-eligible patient profile than would be the case in other parts of the country."

"Even if it were made clear that many physicians justifiably derive a significant part of their income from services rendered to patients covered by Medicare, the figure—though it may appear sizable—will in no way represent the physicians' net income. The most current figures available show physician overhead averaging 40 per cent of gross income, with general practitioners, family physicians, and pediatricians supporting operat-

ing expenses even greater than 40 per cent.

Rep. Thomas Luken (D-Ohio) has also labelled as "objectionable" the HEW list. In a letter to Secretary Califano, Luken said the disclosure "could be deceptive, expensive, and open to a significant rate of error!" The liberal Democrat, a member of the House Commerce Committee, said that Medicare payments often are submitted to individual physicians on behalf of a hospital or clinic. "On such occasions, the HEW listing will not designate the specific physician who worked with Medicare patients. As a result, the list could be deceptive, and would not offer a clear comparison of the actual payments received by the individual physician," said Luken. He told Califano that "it has been estimated that this undertaking will cost the federal government from $750,000 to $1 million, and some $300,000 annually thereafter. Moreover, additional expense must be borne by the carriers, such as Blue Shield.

Luken concluded: "I am not opposed to public disclosure of the use of federal moneys. Yet, in considering the possible inaccuracies, imprecise reporting, and expense associated with this project, I respectfully request your reconsideration of this proposal."

Trace Elements

(Continued from page 186)

essential role in man as well. Secondly, the use of a closely controlled, "trace element-free" environment in these "new" trace element studies is in some aspects a good model for the present trend of highly developed societies toward a cleaner environment, including cleaner, more chemically-defined foods. Experiments have demonstrated that growth and well-being of experimental animals raised under such conditions depends on the presence of environmental contaminants yet unidentified.

Thus, *our knowledge of essential trace elements is not complete but the consumption of a varied diet from as many food sources as possible* can be assumed to meet our requirement for essential micronutrients, inorganic and organic. Realizing the gaps in our knowledge of trace element requirements, the best and only advice that can be given to assure adequate trace element nutrition is to consume a varied, mixed diet.

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TELEPHONES FOR THE DEAF

While no one is certain of the exact number, statistics from the Department of Health, Education and Welfare indicate there are perhaps two million Americans who are deaf or very hard of hearing. Certainly their most serious problem is communication.

Only a minority of this group are able to lip-read or to speak orally. Even so, they still are not able to use a telephone to make a simple call across town.

A small fraction of deaf people solve this problem by purchasing a surplus teletype with a telephone coupler so that they can send typed messages to one another. But these machines are prone to break down and are quickly becoming scarce. Worst of all, they are too large and heavy to be carried on a trip or during daily travel.

In order to help solve this problem, SRI International in Menlo Park, California, is currently developing a pocket-sized, low-cost terminal to enable deaf people to place telephone calls from any telephone anywhere in the country to another telephone having a compatible device.

This battery-operated handheld terminal will be approximately the size of a calculator, and will slip on to the standard telephone handset.

To use the device, the telephone connection is established as usual. Then the two parties can hold a conversation by typing messages on a small keyboard, reading the reply on a small display.

Hospitals, police stations, doctors' offices, libraries, and the homes of relatives are just a few of the places a deaf person would be able to contact with such a device. Some 911 emergency dispatch centers already have teletypes available so that deaf people can obtain emergency services if they have a compatible terminal.

SRI International has just begun development of this device under a grant from the Department of HEW. The handheld terminal for the deaf is expected to reach the public as a commercial product in two-three years. A major design objective is to make the device affordable by essentially everyone.

SRI International, formerly Stanford Research Institute, is an independent, nonprofit organization that performs contract research for worldwide clients in business and government.

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Solution

(Continued from page 223)

were available to him by that magic formula. No one would say Joe shouldn't have this or that service—as long as it was covered. If it meant going to the hospital, it might mean a pretty big bill but no problem—it was covered.

Somehow, though, it wasn't the same with Doc and Joe. More and more this third man in the room helped decide what was wrong and what was to be done about it—after all, as he pointed out, he was paying the bill. It became easier for Joe to figure things should turn out all right and if they didn't, it had to be someone's fault and he allowed that someone had to be Doc. Since Doc couldn't make guarantees, he tried to cover all the possibilities by ordering all the tests he could think of (especially if the policy had a lab rider) before the local plaintiff's lawyer thought of them. This made the insurance man a little testy—fatigue, you know, from staying up late nights trying to figure out the premium increases—not Joe's—Doc's. Still the home office screamed about the money the company was losing and there wasn't much agreement except that it was Doc's fault. And besides, the baseball team was having a losing season too.

So, as it often is with the human experience, the actions to make this a better world, the progressive maneuvers designed to bring health and security within the reach of the Joes—in fact, stuff it down their throats—have brought new problems, as they tried to solve the old ones, in about a three to one ratio.

Well, everyone complains about the situation. But we can't get rid of Joe or Doc and that's because of the last remaining vestige of their relationship—they need each other. So what if we excise the fiscal interloper? Doc goes back to charging Joe what he sees fit and hopes he gets it. He gets rid of all the things he has been complaining about—fee hassles, paper work (some of it anyway), threats to confidentiality, the outsider looking over his shoulder. As he has proclaimed on more than one occasion, he can get back to practicing medicine. Joe should be at least as happy. The boss will boost his pay, of course, since they don't have to hold out for insurance, and Joe can know the joy of developing his character by budgeting so as to be able to pay Doc for what he does do, which won't be as much as it was since it isn't covered (which will make surprisingly little difference in the outcome). Since they will be back on their old buddy basis, Joe won't feel compelled to sue Doc for loss of his manhood after the rupture operation (another baby being on the way anyway). The insurance company will be happy. They'll stop losing all that money they have been talking about and return to the simpler life of burgling, auto accidents, and hail damage. And they can go ahead and buy that movie company they've been dickering for.

But most of all, Doc and Joe will be back in their seats across the desk from each other—no one else in the room—pondering what they learned from their experience and wondering how long this strange feeling of having found each other will last.

But then the whole idea's ridiculous, isn't it?—*D.E.G.*

NEW MEMBERS

The JOURNAL *takes this opportunity to welcome these new members into the Kansas Medical Society.*

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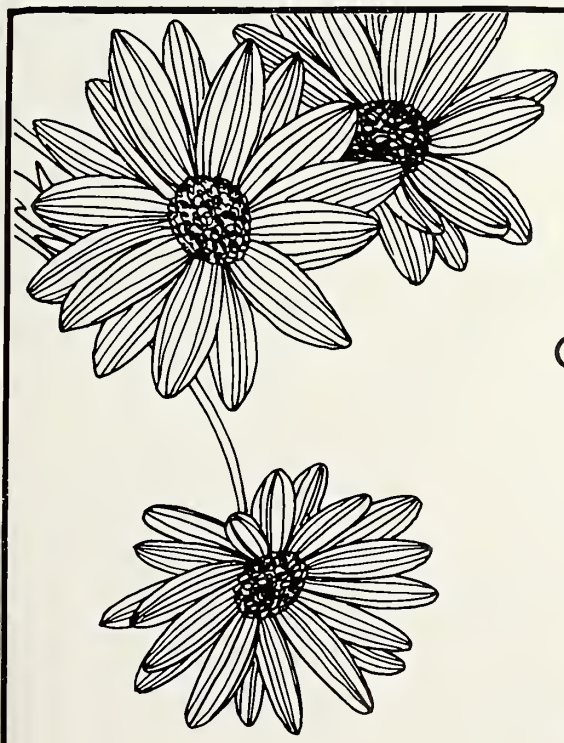
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Composition: Vasodilan tablets, isoxsuprine HCl, 10 mg. and 20 mg.

Vasodilan injection, isoxsuprine HCl, 5 mg., per ml.

Dosage and Administration: Oral: 10 to 20 mg., three or four times daily.

Intramuscular: 5 to 10 mg. (1 or 2 ml.) two or three times daily. Intramuscular administration may be used initially in severe or acute conditions.

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Administration of single dose of 10 mg. intramuscularly may result in hypotension and tachycardia. These symptoms are more pronounced in higher doses. For these reasons single intramuscular doses exceeding 10 mg. are not recommended. Repeated administration of 5 to 10 mg. intramuscularly at suitable intervals may be employed.

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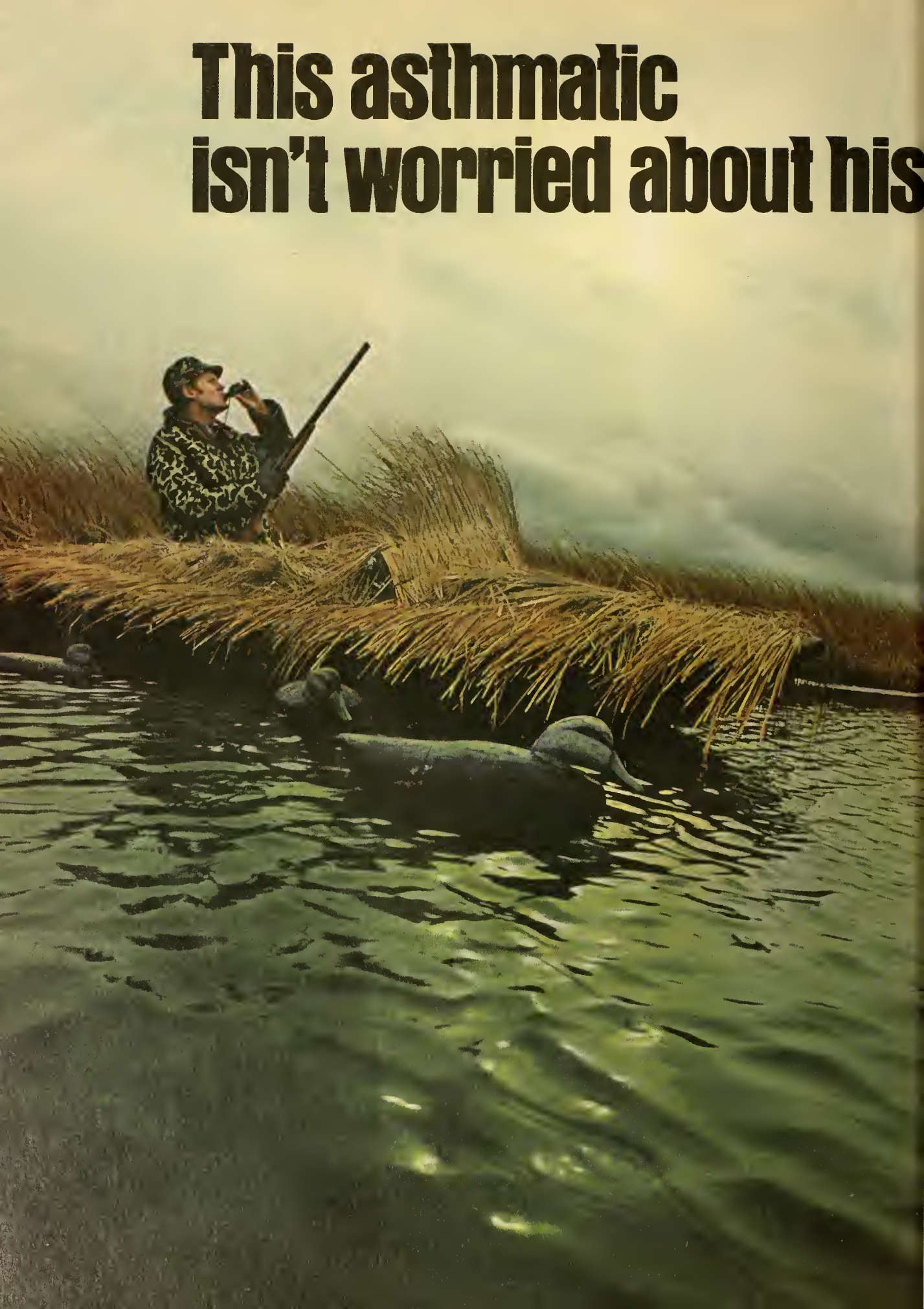
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Now, for the asthmatic who requires high-dose theophylline therapy for therapeutic serum concentrations

Mead Johnson Pharmaceutical Division announces

QUIBRON®-300

Each capsule contains 300 mg theophylline (anhydraus) and 180 mg glyceryl guaiacalate (guaifenesin)

For Brief Summary, please see the last page of this advertisement.

QUIBRON®-300

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The new high-dose theophylline capsule... for dependable theophylline therapy when products of lower dosage do not adequately control asthma symptoms.

Specially formulated

...for optimal efficacy

Quibron-300 is appropriate therapy for asthma patients whose symptoms are not adequately controlled on lower doses of theophylline, particularly for patients whose theophylline dosage has been adjusted upward to achieve therapeutic serum levels. In one study,[1] an average peak increase in FEV_1 of 35% was demonstrated after a single dose equivalent to one Quibron-300 capsule, and significant improvement in this pulmonary function lasted for nearly eight hours after administration.

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Adverse reactions to theophylline are related to serum levels and are usually not a problem at concentrations below 20 mcg/ml. Of 45 patients studied[1] after a single dose, only seven reported adverse reactions. The most common reaction was a feeling of lightheadedness by three of these seven patients.

Reference 1. Data on file. Mead Johnson Pharmaceutical Division.

Indications: For the symptomatic treatment of bronchospastic conditions such as bronchial asthma, asthmatic bronchitis, chronic bronchitis, and pulmonary emphysema.

Dosage: Quibron—Adults: 1-2 capsules or 1-2 tablespoonfuls elixir every 6-8 hours. Children under 12: 4-6 mg theophylline/kg body weight every 6-8 hours.

Quibron-300— Adults: 1 capsule every 6-8 hours.

Theophylline dosage may be cautiously increased to 2000 mg/24 hour in adults and 9 or 10 mg/kg every 6 hours in children. Monitoring of serum theophylline levels at higher dosages is recommended.

Precautions: Do not administer more frequently than every 6 hours, or within 12 hours after rectal dose of any preparation containing theophylline or aminophylline. Do not give other xanthine derivatives concurrently. Use in case of pregnancy only when clearly needed.

Adverse Reactions: Theophylline may exert some stimulating effect on the central nervous system. Its administration may cause local irritation of the gastric mucosa, with possible gastric discomfort, nausea, and vomiting. The frequency of adverse reactions is related to the serum theophylline level and is not usually a problem at serum theophylline levels below 20 μg/ml.

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Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

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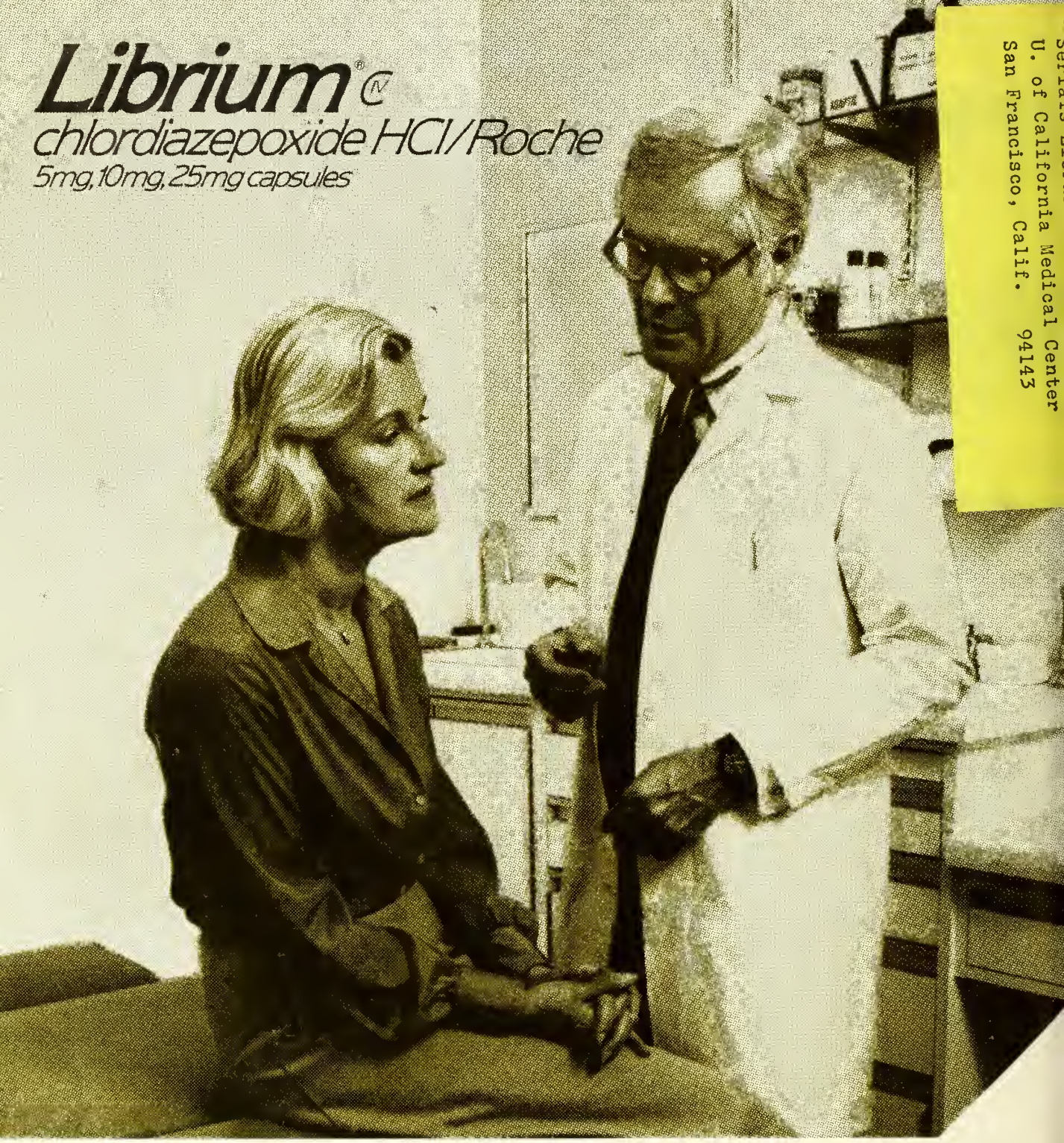
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JUNE
1978

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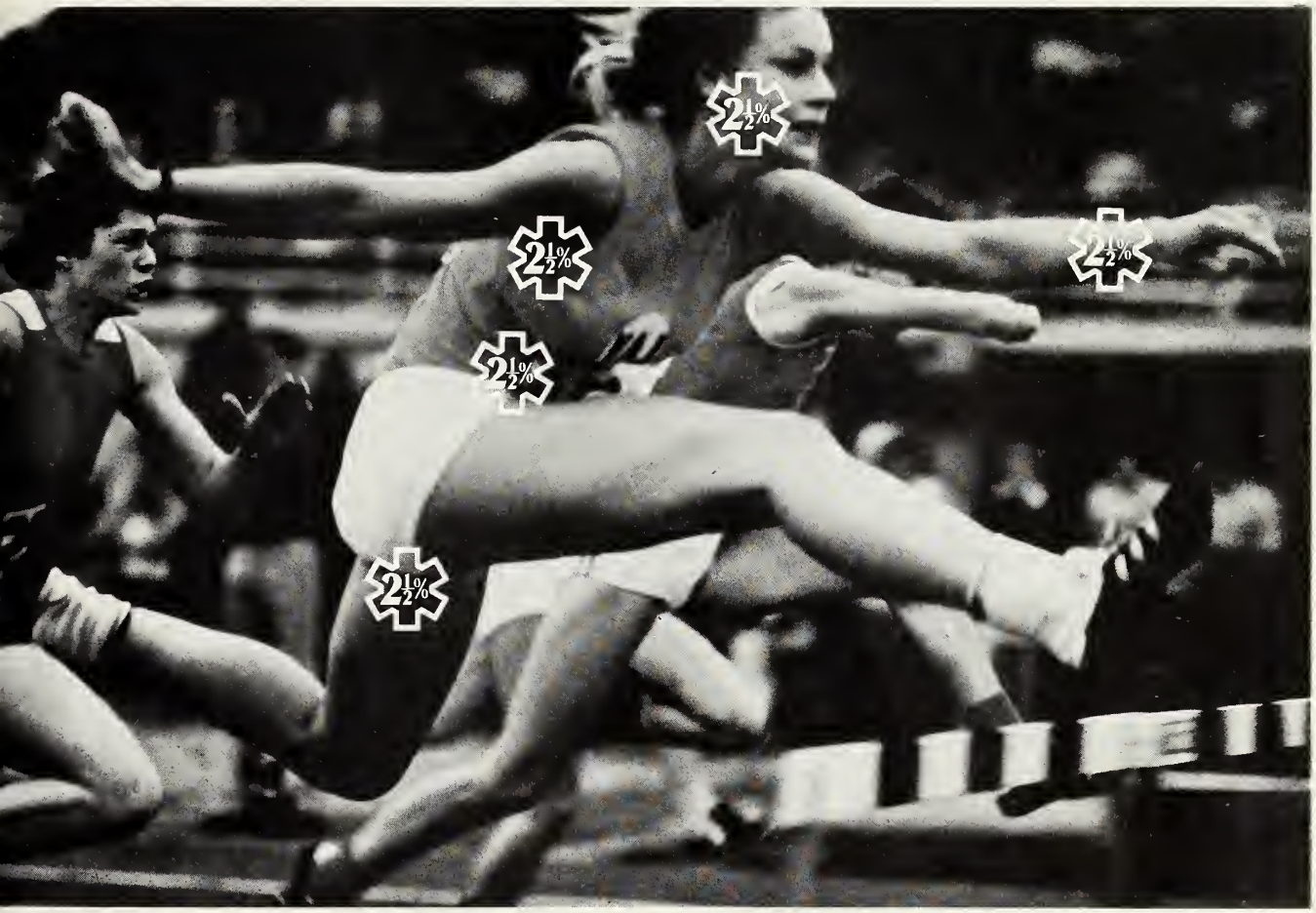
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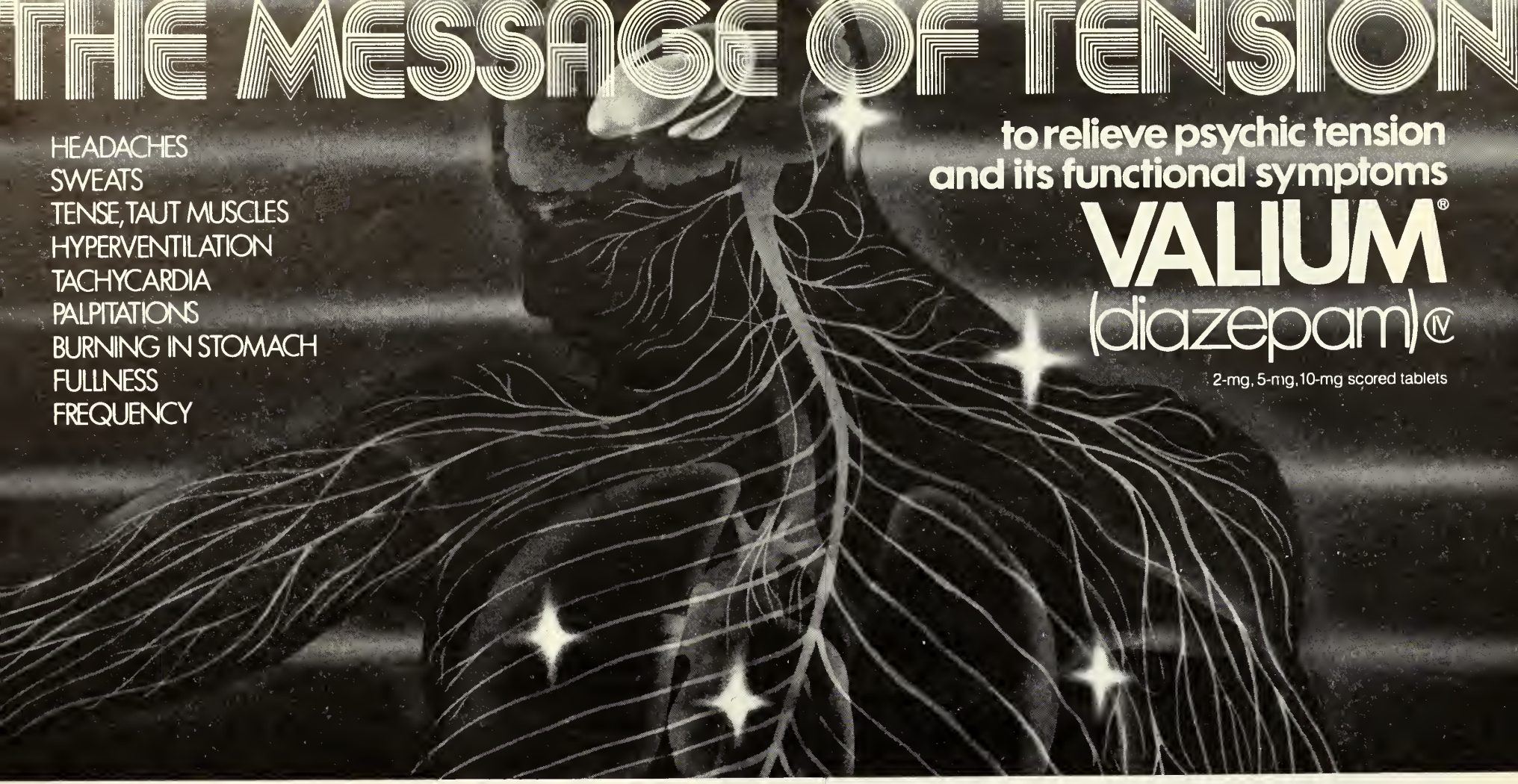
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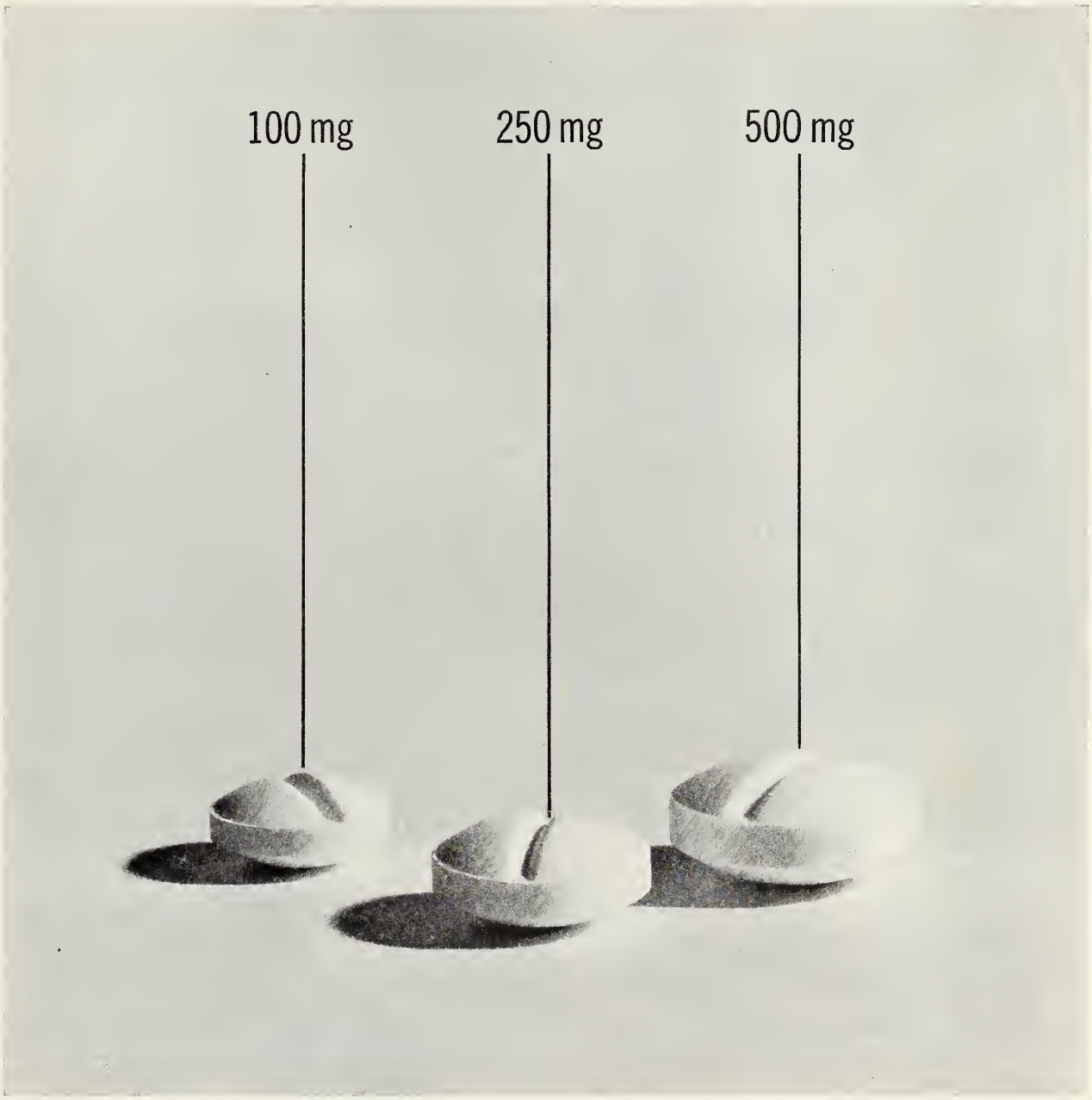
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Book REVIEWS

THE COMPULSIVE OVEREATER: Seven Steps to Thin Sanity, by George F. Christians, Doubleday & Company, Inc., New York. 1978. 120 pages. $5.95.

Prescribe a regimen of one shot of bourbon tid for an alcoholic and what would happen? The idea seems too ridiculous to consider; yet this is analogous to the dilemma of the compulsive overeater, for food, unlike alcohol, is essential to life. With this concept, George Christians enlisted the sympathetic aid of a recovered alcoholic physician in his life-long battle with obesity—an obesity which, like most, was caused not by glands, not by heredity, but simply by eating too much food. Together they worked out the details of a plan which has enabled the author to achieve and maintain a near-normal weight.

Mr. Christians does not believe in "diets." He contends that while adherance to a specific diet may result in a loss of weight, the loss will not likely be permanent because the basic problem—*i.e.,* compulsive overeating—has not been addressed. He correctly points out that three balanced meals per day with a total caloric value equal to the maintenance requirement for a person's desirable weight will result in a weight loss—possibly not rapid or dramatic, but steady and sure.

He believes that compulsive overeating—like alcoholism—is never cured, but can be controlled. He strongly emphasizes the one-day-at-a-time concept.

His seven steps to thin sanity are:

(1) Admit to yourself and to others that you are a compulsive overeater.

(2) Eat a normal maintenance diet of three balanced meals per day with a total caloric value necessary to maintain correct weight.

(3) Learn to be thin by associating with other "fats" who will allow you to assume a thin role when you succeed in becoming a normal thin.

(4) Resolve to live (and eat) one day at a time—each with three nutritious, balanced meals planned in advance with nothing between except low calorie beverages.

(5) Handle other problems on a one-day-at-a-time basis, also. (Mr. Christians brings in the concept of God here, but suggests reliance on any personal inner resource from which strength and serenity can be derived.)

(6) Love yourself as you would expect others to love you.

(7) Include regular daily exercise as part of your program.

The book is short and easy to read. Its concepts are not startling or revolutionary, yet they combine to suggest a workable procedure by which the compulsive overeater may bring the problem under permanent control. —*E.B.*

MEALTIME MANUAL for People With Disabilities and the Aging, Second Edition. Compiled by Judith Lannefeld Klinger, O.T.R., M.A., with the Institute of Rehabilitation Medicine, New York University Medical Center, and Campbell Soup Company. (Order from Mealtime Manual, Box 38, Ronks, PA 17572.) 269 pages. $3.25.

"Just as men and women everywhere yearn to be free, so do they want to be as self-sufficient and productive as possible. Homemakers with disabilities are no exception." This fundamental assertion from the foreword by Howard Rusk, M.D., delineates the guiding concept of this excellent book. It is a recipe book of sorts, but even a cursory examination reveals a much broader scope.

The first 50 pages are crammed with practical hints for persons with various specific physical disabilities. Generous use of illustrative photographs; definite information on helpful accoutrements, their price and source; and numerous references for locating additional help and information in specific problem areas—all of these combine to make this section alone invaluable to a newly disabled person struggling with frustration and despair resulting from physical limitations.

(Continued on page 263)

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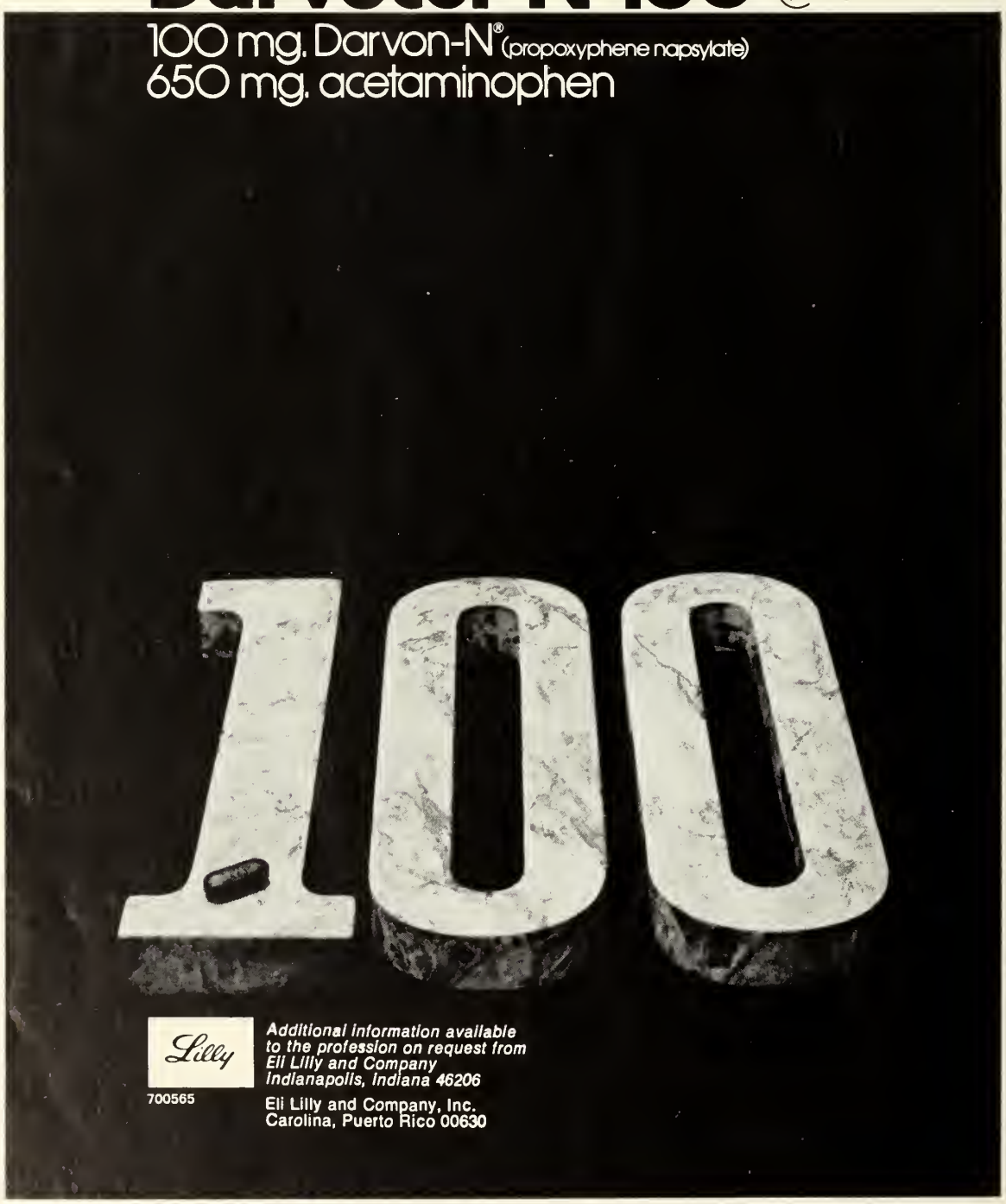
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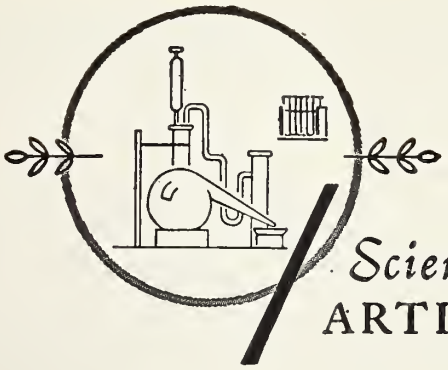


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Posterior Urethral Injuries

Procedural Evaluation and Management

JOHN W. WEIGEL, M.D., DAVID STEWART, M.D. ***and***
ROBERT FOSTER, M.D., ***Kansas City, Kansas***

Posterior urethral injuries may occupy a low priority for the severely traumatized patient; however, improper initial therapy can result in considerable morbidity. Causes of injury, and diagnostic and therapeutic procedures are discussed and evaluated.

POSTERIOR URETHRAL INJURIES can occur with instrumentation, perineal surgery (*e.g.,* A-P resection of carcinoma of the rectum), gunshot or knife wounds, straddle injuries, and pelvic fractures. Recognition and proper management are essential to avoid the considerable morbidity which often accompanies such an injury. Potentially the most serious form of posterior urethral injury is the prostate-membranous urethral disruption which accompanies pelvic fracture in seven per cent of cases.[1, 2]

Causes

The mechanism of injury of the posterior urethra with instrumentation, surgery, knife or gunshot wounds is obvious. Straddle injuries are responsible for 15-30 per cent[3, 4] of posterior urethral injuries and result from direct perineal trauma. Pelvic fractures associated with urethral injuries tend to involve the pubic rami and symphysis, are multiple, and are associated with displacement. There is a high association of laceration of the puboprostatic ligament. The urethral injury is usually due to force producing soft tissue disruption, rather than to laceration by a spicule of bone.[5]

Diagnosis

A urethral injury should be suspected when a patient cannot void, exhibits blood at the urethral meatus, or presents with a pelvic fracture (particularly if the fracture is multiple or associated with displacement). Rectal examination may reveal bogginess of the prostate in the usual prostatic position. Digital detection of upward displacement is not common. Confirmation of the extent of urological injury can be obtained with intravenous pyelogram and retrograde urethrogram. With complete urethral rupture, upward displacement and "tear drop" deformity of the bladder may be seen. The urethrogram will demonstrate extravasation of contrast with urethral injury. Extravasation with concomitant retrograde flow into the bladder characterizes partial urethral tears.[6]

Therapy

It is imperative that such an assessment of possible urethral injury be made prior to catheterization, since an inexpert attempt may result in additional urethral trauma by extending a urethral tear.[7] Urethral injuries occupy a low priority for the severely injured patient, but the tendency to insert a catheter to monitor urine output should be overcome because other indicators—blood pressure, progressive resistance, central venous pressure—

From the University of Kansas Medical Center, Kansas City, KS.

can guide fluid resuscitation. Therapy should be aimed at minimizing the complications associated with urethral injuries and their repair, namely: stricture, incontinence, and impotence. The controversies in treatment primarily involve a catheter stint vs. no catheter, and immediate vs. delayed urethral repair. Most reasonable management would seem to argue against a catheter stint, particularly in those cases where the urethra would be expected to heal without an operation. Insertion of a catheter could —even in expert hands—extend a partial tear, additionally traumatize the urethra, and impede free drainage of exudates.[7] Immediate primary repair has been associated with good results in some series[8] but not in others.[3, 7, 9] Delayed repair avoids operations on acutely traumatized tissues in which tissue planes are destroyed by edema or hematoma. Use of the Johonson technique after a delay of three-four months has allowed good results for urethral injuries requiring realignment.

Summary

The best results seem to be obtained with cystostomy drainage for 10-14 days for minimal injuries (those with partial urethral tears without displacement). Best results for more extensive injuries could be expected with cystostomy drainage and delayed repair using the Johonson technique.

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Unusual Complications

Report of a Case of Appendicitis

W. G. CAUBLE, M.D., *Wichita*

THE DIAGNOSIS of appendicitis can sometimes be quite difficult and perplexing. Good physical examination and history are of utmost importance, and there is nothing that can replace these two procedures. Nevertheless, the history sometimes does not give helpful information and the physical examination may be questionable. As with the case presented, the primary diagnosis may be secondary to appendicitis, and this fact should be kept in mind when dealing with right lower quadrant pain and tenderness, evidence of abscess formation, urinary disease, or even right thigh symptoms.

Richie *et al.*[1] reported three patients with appendicitis presenting respectively with bladder tumor, hematuria, and pelvic mass with ureteral obstruction. Barium enema revealed an appendicovesical abscess in the patient with the mass. In the second case, intravenous pyelogram revealed a possible bladder tumor; the patient had gross hematuria. A right pelvic mass with drainage into the vagina was found in the third patient. Sampson *et al.*[2] reported a case with a large abscess of the right thigh involving the right psoas muscle. This led to a gangrenous retroperitoneal appendix. The patient subsequently died. Murray and Molavi[3] reported a case of right perinephric abscess caused by acute appendicitis. The abscess was subsequently drained, and the dye injected into the abscess cavity appeared in the colon. This occurred after a barium enema was reported negative (it had been drained previously). Milewski[4] reported a case in which the appendiceal stump eroded into the cecum and caused severe bleeding.

Case Report

A 22-year-old white male was referred by an internist. The patient presented with a large painful mass in the right groin of nearly three weeks duration. He gave no history of any indigestion or discomfort in the abdomen. His bowels had been functioning normally. Under general anesthesia, the mass was found to be an abscess and was drained. No sinus tract of any kind was found. A barium enema produced negative results. The patient did well and the drainage subsided. A month later more drainage occurred; the abscess reoccurred later, was drained, and an opaque dye was injected into a sinus area which was found at that operation. This revealed a rather long fistula tract extending to the pericecal area, with a fecalith occluding the distal end of the appendix *(Figure 1)*.

Literature relating to unusual complications of appendicitis is reviewed, and an interesting case history, involving no intestinal complaints, is presented.



Figure 1. Sinus tract demonstrated running from the right groin abscess to the appendix area.

Address reprint requests to Dr. Cauble, 1148 South Hillside, Wichita, KS 67211.

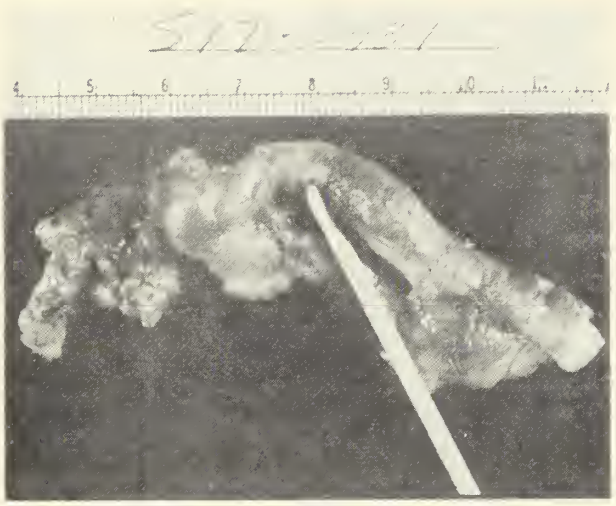


Figure 2. Appendix with small perforation which was the source of the fistula.

The patient's numerous adhesions were lysed and an appendectomy was performed. The appendix was pressed against the right side of the pelvis and was covered with the omentum. A fistula led from it to the right groin, and the appendix revealed a small perforation *(Figure 2)* which was the source of the fistula. The postoperative course was uneventful and the patient has been free of complaints for nearly a year.

Summary

Unusual complications of appendicitis have been discussed and some of the literature reviewed. A case of acute appendicitis was presented which had no intestinal complaints but involved a large abscess in the right groin. A fistula tract was discovered running to the appendix. Appendectomy cured the patient and he has had no symptoms for several months.

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A Comparison

Scratch Testing and RAST in the Allergic Evaluation

ARNALDO CAPRILES HULETT, M.D., DAVID L. STRAUB, M.D., MARJORIE TRINKL, R.N., P.N.A., JAMES A. CAPLIN, M.D., KENT DEYARMAN, M.D., JAMES B. NEIBURGER, M.D., BRUCE L. PFUETZE, M.D. *and* ROBERT J. DOCKHORN, M.D.,

Kansas City, Kansas

THE LITERATURE contains many reports regarding the usefulness of the radioallergosorbent test (RAST) in the field of allergy. Among other uses,[1–3] it has been employed in diagnostic allergy, and its results compared to provocation and skin testing.[4–12] Most of the studies have involved the intracutaneous (intradermal) technique, puncture (prick) test, or both. Skin tests performed by the scratch method, although widely used by practicing physicians throughout the United States, have never been extensively compared to the RAST.[13] Following is the report of our experience with RAST and skin testing by the scratch technique in a large number of children seen in our allergy clinic.

Materials and Methods

Randomly selected from the children seen between January 1974 and January 1976, 335 were included in the study. Their ages ranged from 1-18 years, with a mean of 7 years.

Specific IgE antibodies for 12 allergens were determined in the patients' serum using a commercially available method with filter paper as solid phase. As suggested by the manufacturer, the results were reported on a scale of 1+ to 4+. For the purpose of analysis, scores of 1+ or above were arbitrarily taken into account.

The 12 antigens considered important in the Kansas City area that were assayed included: perennial rye grass, timothy, common ragweed (short), English plantain, elm, cat epithelium, dog epithelium, cladosporium herbarum, alternaria tenuis, egg white, milk, and house dust.

As part of the patients' allergy evaluation, scratch tests were performed on the back with a Berkeley scarifier. Allergen extracts at 1/10 dilution weight by volume (w/v) were used. Glycerin 50 per cent served as a negative control, and histamine 1/1000 w/v was used as a positive control.

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Scoring of skin reactivity was done as shown in *Table I*. It was decided for the purpose of analysis, to compare the RAST results with 1+ or greater skin test scores and then only with scratch test with 2+ or above.

A comparison between scratch skin tests and the radioallergosorbent test was made in 335 patients seen over a period of two years during routine allergy evaluations. Results indicate that scratch testing alone is not an adequate measure of the patient's allergic status.

Results

A total of 3996 RAST tests were performed on our population of 335 patients. These results *(Table II)* are analyzed for each antigen and correlated with the skin test results. The first column shows the agreement between RAST and positive skin tests. The second column considers the incidence of positive RAST in patients with negative skin tests. In search of a better agreement to RAST, only skin tests scored at 2+ or greater were taken into account for comparison, as

TABLE I

Negative ..	No reaction or a reaction no different from the control.
1+	Erythema less than a nickel in diameter (21 mm).
2+	Erythema larger than a nickel in diameter.
3+	Erythema and wheal formation without pseudopods.
4+	Erythema and a wheal with pseudopod formation.

TABLE II

AGREEMENT BETWEEN RAST AND SCRATCH TESTS EXPRESSED AS PERCENTAGE OF NUMBER OF SUBJECTS WITH POSITIVE RAST

Allergen	*RAST (+) / Scratch (+)*	%	*RAST (+) / Scratch (−)*	%	*RAST (+) / Scratch (2+)*	%	*Scratch (+) / Total No. of Patients*	%
Elm (Ulmus americana)	34/93	36.5	32/242	13.2	31/67	46	93/335	27.7
Ragweed (Ambrosia elatior)	60/150	40	108/185	32	52/108	48.1	150/335	44.7
English plantain (Plantago lanceolate)	26/66	39.4	43/269	15.9	24/58	41.4	66/335	19.7
Grasses*	56/147	38	35/178	19.6	50/120	41.6	147/325	45
Alternaria tenuis	76/152	50	43/183	23	69/119	57	152/335	45
Cladosporium herbarum	30/150	20	18/185	9.7	26/117	22	150/335	44.5
Cat epithelium	32/87	36.7	57/248	22.9	24/53	45	87/335	25.9
Dog epithelium	23/105	21.9	23/230	10	12/59	20.3	105/335	31.3
Eggwhite	51/71	71	34/261	13	13/41	31.7	71/332	21.3
Milk	9/55	16.3	21/279	7.5	5/27	18.5	55/334	16.4
Housedust (Greer Labs)	33/112	29.4	41/223	18.3	23/73	31.5	112/335	33.4
Mean		25.6%		16.8%		36.6%		32%

* The grasses, perennial rye grass (Lolium perenne) and timothy (Phleum pratense), are grouped together due to identical results.

shown in the third column. The overall incidence of skin reactivity as well as that for each allergen can be seen in column four.

Discussion

In the report by Apold *et al.*,[13] skin tests by scratch, prick, and intradermal methods were compared to RAST in the diagnosis of reaginic allergy. The agreement between these procedures is difficult to assess because all skin tests were analyzed as a group. A comparison of results by scratch skin tests and the commercially available RAST is presented below.

As seen in *Table II*, the overall agreement between RAST and scratch test results (first column) is less than 30 per cent. There was a 71 per cent agreement with egg white antigen. All other correlations were 50 per cent or less. When 2+ or greater scratch test results (third column) were considered, the mean agreement improved approximately 10 per cent.

The second column demonstrates an important point. There is a mean incidence of nearly 17 per cent of pa-

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Pulmonary Microembolism

Respiratory Failure and Pancreatitis

NIRADON WONGSURAWAT, M.D., SALVADOR DIPALING, M.D., *and*
VANEE WONGSURAWAT, M.D., *Topeka*

EARLY RESPIRATORY insufficiency is a frequent and potentially lethal feature of the initial phase of acute pancreatitis.[1] Recognition of the pulmonary complication and aggressive therapy may improve the eventual outcome. The correct diagnosis must, of course, be made before appropriate therapy can be instituted. We present here a case of respiratory failure without obvious clinical or radiographic pulmonary signs where acute pancreatitis was diagnosed at autopsy.

Case Report

A 61-year-old male was admitted to the Medical Service in a state of severe distress. Because a laryngectomy for cancer of the larynx had been performed two years previously, communication was difficult and it was not possible to obtain a detailed history. There was no history of diabetes mellitus or previous pancreatitis.

The patient was in poor general condition and severely dehydrated. His pulse rate was 132/min; blood pressure, 130/90 mm Hg; respiration rate, 26/min; rectal temperature, 38.8C. Bilateral basal rales were audible on chest auscultation. There were considerable epigastric tenderness and diminished bowel sounds. Radiographic examination of the chest yielded normal results. Arterial blood gas values were: pH 7.41, pO_2 55.2 mm Hg, and pCO_2 25.8 mm Hg. Lung scan showed areas of perfusion defect on peripheries of both lower lungs. Significant laboratory findings were: hemoglobin, 20.5 gm/100 ml; white blood cell count (WBC), 17,800/cu mm with 90 per cent polymorphonuclear cells; blood glucose, 534 mg/100 ml; calcium, 11.1 mg/100 ml; phosphate, 6.5 mg/100 ml; total serum protein, 8.3 gm/100 ml; albumin, 4.7 gm/100 ml. Urinalysis showed 4+ glucose, 3+ protein, and 3+ ketones.

The clinical diagnosis included overt diabetes, pulmonary embolism, and probable pneumonia. Therapeutic regimen consisted of intravenous fluid, intravenous and intramuscular regular insulin, intravenous heparin, and antibiotics. The patient's condition deteriorated rapidly. Rectal temperature rose to 41.4C; respiration rate, 36/min; pulse rate 140/min. Serum amylase measured several hours after admission was 1400 units/100 ml (Somogyi). The patient died nine hours after admission.

Respiratory insufficiency, frequently seen early in the course of acute pancreatitis, is an important cause of the associated morbidity and mortality. A case of acute hemorrhagic pancreatitis complicated by respiratory insufficiency is presented and the pathogenesis discussed.

Postmortem examination revealed acute hemorrhagic pancreatitis *(Figure 1)*. The lungs were markedly congested with extravasation of red blood cells into the alveoli *(Figure 2)*. Fibrin clots in pulmonary arterioles were detected *(Figure 3)*. Parathyroid glands were searched for but, due to fibrosis secondary to laryngectomy, none of the parathyroid glands were identified.

Hypercalcemia was detected in our patient. There was no evidence of recurrence of laryngeal cancer. The possibility of bone metastasis cannot be ruled out even though it is uncommon.[2] Hyperparathyroidism was another possible cause of hypercalcemia in this patient. It is questionable, however, that this mild hypercalcemia was the cause or one of the causes of acute pancreatitis.

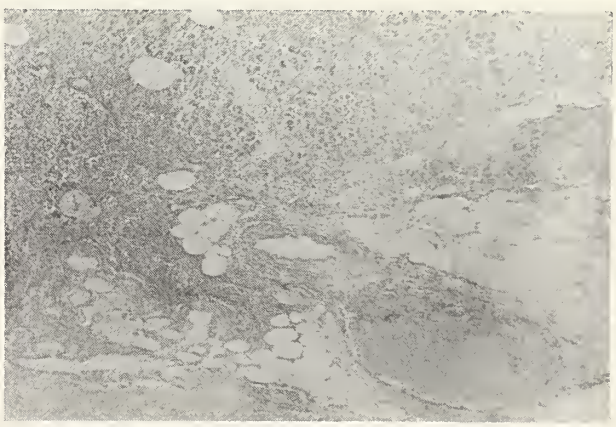


Figure 1. Acute hemorrhagic pancreatitis.

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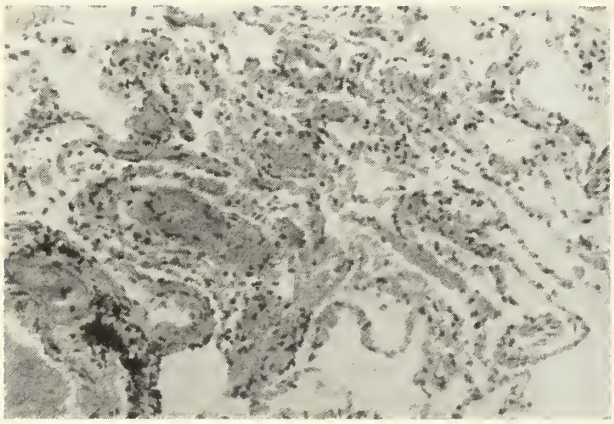


Figure. 2. The lung showed severe congestion with extravasation of red blood cells into the alveoli.

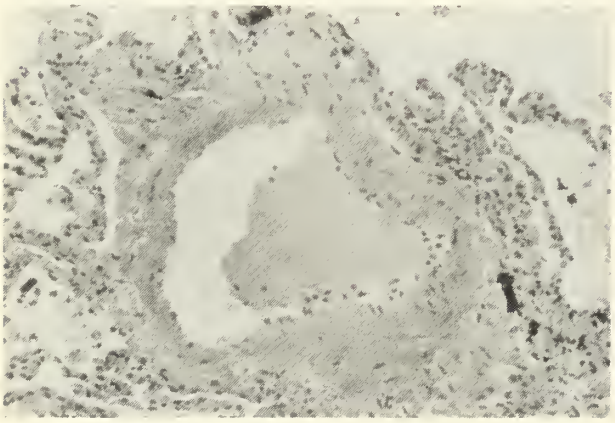


Figure 3. Microembolism in pulmonary arteriole.

Comment

Acute pancreatitis in our patient was not considered until much later. The result of serum amylase was not available until after the patient's death. Based on nonspecific findings—dyspnea, chest pain, hypoxemia, hypocarbia, perfusion defect in the lung scan, and normal chest roentgenogram—pulmonary embolism was erroneously interpreted as the primary illness.

The pulmonary manifestations associated with acute pancreatitis have only recently been recognized.[3] Of 84 patients studied, 45 per cent developed arterial pO_2 levels below 60 mm Hg at some time during the first week of illness.[4] The early phase of respiratory insufficiency complicating acute pancreatitis is frequently apparent only by arterial blood gas measurements. In the later phase, obvious clinical and radiographic pulmonary signs become evident.

The pathogenesis of respiratory complication following acute pancreatitis remains a subject of controversy. The factors most commonly implicated are abdominal distension, pain and guarding with retroperitoneal edema and elevation of the diaphragm, and presence of enzyme-rich pleural effusions.[5-8] Ventilatory function studies during the early phase of pancreatitis have demonstrated a decrease in pulmonary compliance and an increase in resistance.[9] Experimental studies in dogs have indicated a marked increase in venoarterial shunting, an increase in minute respiratory volume and oxygen consumption, and a decrease in the lung stability index following the induction of acute pancreatitis.[10, 11] Circulating pancreatic enzymes may play a role in these respiratory complications. Elevated serum levels of pancreatic lecithinase have been documented in patients with acute pancreatitis.[12] Lecithin is a major component of pulmonary surfactant, and intravenous infusion of a lecithinase has resulted in respiratory distress and alteration of pulmonary surface properties in dogs.[13] However, no significant changes in pulmonary surfactant or in circulating lecithinase were detected in experimental acute pancreatitis.[10]

The role of intravascular coagulation and thromboemboli involving the pulmonary microcirculation in the pathogenesis of the respiratory distress syndrome has been emphasized.[14] Alterations in coagulation factors have been documented during acute pancreatitis;[15] fibrin the induction of experimental acute pancreatitis.[16] Fibrin clots were found in pulmonary arterioles in our patient. It is possible, therefore, that while pancreatic enzymes may have a precipitating role in respiratory complications of acute pancreatitis, the final pathogenesis is related to pulmonary microembolism.

The progression of the pulmonary failure can often be reversed by methods that improve ventilation.[15] Support of ventilation by mechanical measures, using either volume cycle or pressure-triggered respirators, may be indicated. The use of a volume ventilator has a splinting effect on the alveoli and ultimately results in better ventilatory function.[1] If the pO_2 cannot be stabilized, positive and expiratory pressure is indicated. The use of diuretics alone or in combination with albumin has been found to be effective.[7, 17]

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Special Series—III

Frozen Red Cell Transfusions

ANTONIO HUAMAN, M.D.,* ***Topeka***

BLOOD TRANSFUSIONS contribute to the success of modern surgery, emergency medicine and oncologic treatment; however, its widespread use has caused some problems such as the development of abnormal antibodies, transfusion reactions, and the ever present threat of unavailability when blood is needed on short notice. It is true that the establishment of blood banks has solved many problems in blood supply, but the system is not perfect because—due to its innate characteristics—blood cannot be produced, controlled, and distributed as are manufactured drugs. An important limitation of transfusion blood is its utilization span of 21 days.

Efforts to overcome this time limit have ranged from modification of the anticoagulant solutions to preparation of synthetic red cells. While the solutions extend the usable shelf life of blood up to 28 days, the preservation of red cells by freezing techniques may render the artificial red cells unnecessary.

The preservation of red cells by freezing was an outgrowth of the techniques for preservation of spermatozoa by Polge, *et al.*[1] This is made possible by the utilization of certain chemical compounds called endocellular cryophylactic agents (ECA), which bind tightly to the intracellular water and protect living cells at low temperatures by preventing orderly ice crystallization. One of these agents, glycerol, at a concentration of 4.8 m-5.5 m, allows the freezing and thawing of erythrocytes with only minimal hemolysis. The glycerol must, however, be removed before transfusion. Techniques to separate glycerol from water have been unsuccessful because of the tight water-glycerol bonding, but methods such as dilution, dialysis or centrifugation, that remove glycerol and water as a complex, have proved more effective.

Huggins[2] succeeded in removing glycerol from thawed red cells by reversible agglomeration using non-electrolytic solutions of dextrose and fructose. He designed the methods for glycerolization, freezing, and deglycerolization of red cells for the most successful service of its kind at the Massachusetts General Hospital. Others have used continuous flow centrifugation and computerized centrifugation with equally good results, bringing the methodology for red cell transfusion to a practical and safe status.

Methodology

The preparation of frozen-thawed red cells for transfusion essentially consists of the following phases:

1. glycerolization—a glycerol solution containing non-electrolytes such as fructose and glucose is added to packed red cells which have been previously drawn into ACD or CPD solutions;

2. quick freezing of the red cells is accomplished at –80C, and the frozen blood is then stored at the same temperature for months or years.

3. before transfusion, the frozen red cells are thawed in water bath at 37C;

4. deglycerolization by either reversible agglomeration or centrifugation is carried out with addition of saline and glucose solution. This process can be done by manual methods utilizing the cytoglomerator of Huggins, by continuous flow centrifugation (Haemonetics) or by computerized centrifugation in the IBM cell processor; and

5. the washed cells are dispensed for transfusion as packed cells and must be typed and crossmatched as for conventional transfusions. In addition, the freezing and thawing processes require additional quality control.

Quality Control

Frozen red cell transfusion is not a procedure that can be done on the spur of the moment. It requires adequate physical facilities, equipment, professional competence, and very strict quality control. The latter includes the mandatory elimination of glycerol from the red cells, the elimination of dissolved plasma hemoglobin and potassium from the supernatant, the quantitation of red cell volume, and the prevention of infection. Other parameters that should be worked out before the establishment of the service include the erythrocyte survival time and the determination of 2-3 DPG (diphosphoglycerate) and ATP (adenosine triphosphate). The pro-

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TABLE I

FROZEN RED CELL TRANSFUSIONS

PROTOCOL FOR FROZEN-WASHED RED CELLS

Unit No.: ..... Donor: ..... Freezing No. ..... BLOOD GROUP

Date drawn: ..... Place: .....

Freezing Procedure (IBM) Date: .....

Packed Cell Weight: ..... gm Glycerolyte-57 ..... ml
Bag weighs: ..... gm Initial Vol.: ..... ml
Actual Cell Weight: ..... gm Remaining Vol.: ..... ml

Freezing Control: Adequate .... Defective .... Discarded (Date) .....

Washing Procedure (IBM) Date: .....

Quality Control (Mandatory):	*Limits*	*Quality Control Optional:*	*Limits*
Blood Group & Rh		Coombs Test Direct	Neg.
Hemolysis:	Neg.	Hemoglobin (Supernatant) .... mg%	10mg%
Glycerol ..... m/mols	< 1.0%		
Bacteriology	Neg.	2, 3-DPG ..... uMol/gmHb	> 10.0u
Actual weight of cells ..... gm		ATP ..... uMol/gmHb	> 3.5
Hematocrit .....%	< 70%	Osmotic Fragility	
Extracellular K+ ..... mEq/L	< 1.42mEq/L	Others	

Accepted ..... Rejected ..... Med. Tech. .... Date: .....

Cross match compatible with: ..... Given: ..... Date: .....

12-21-77

tocol used in our institution is illustrated in *Table I.*

According to present published experience,[3, 4] the intracellular glycerol should be less than one per cent because concentrations greater than 1.7 per cent would produce hemolysis during the crossmatch. The supernate hemoglobin after the washing phase should be in the area of 246 mg/unit, or roughly 10 mg/100 ml in the supernatant. The hemoglobin will rapidly increase during the first 24 hrs; therefore, the red cells should be rewashed if blood must be transfused after that time. The extracellular potassium also increases after washing and should be maintained below 1.4 mEq/1.0 ml of blood. Old transfusion bags should be cultured and the results reported to the patient's physician in the shortest time possible, using radiometric bacteriologic methods for fast reporting. The most common bacterium present has been Staphylococcus epidermidis which can remain alive at 4C. The frozen-thawed red cells should be transfused within 24 hours of thawing and washing.

Clinical Applications

Frozen red cell transfusions, in spite of the relatively short time of usage, offer definite advantages over conventional transfusions in the following areas:

1. *Prevention of alloimmunization.* Conventional blood transfusion includes leukocytes, platelets, and materials thereof which are not taken into account, as red cell compatibility is all that is required for transfusion. However, patients who have received many transfusions develop a high incidence of crossmatch incompatibilities, due to leukocyte and platelet antibodies of the HLA type. In frozen red cell transfusions only purified red cells are administered, thereby eliminating the opportunities for the formation of tissue antibodies. This is of significant advantage for candidates of organ transplants, particularly renal transplants, since the lack of sensitization to tissue antigens will decrease the possibilities of rejection of the transplanted organ.

2. *Autologous transfusion.* Frozen red cell transfusions have enhanced the practicality of autologous transfusions in which patients deposit their own blood for use during their own surgery. This procedure is particularly indicated in patients with difficult crossmatch, uncommon blood groups, and for elderly persons who are not

covered by blood bank services. Since prolonged storage of blood cells allows the patient time for blood regeneration between donations, it also facilitates bringing the patient to the operating table in optimal hematologic condition.

Autologous transfusions in our institution have been recently documented by Baker, Lynch and Polly,[5] who reported their successful experience. with 45 patients undergoing surgical joint replacement. Autologous transfusions are also advantagous for patients undergoing coronary by-pass, pneumonectomy, and others who may even be moderately polycythemic and whose blood can only be used for themselves. It is forseeable that more patients will use autologous transfusions, thus alleviating the difficulty found in blood procurement.

3. *Utilization of blood resources.* Frozen red cell transfusions are instrumental in maintaining adequate blood inventories in the blood bank. The increase of the lifespan of transfusable red cells allows for full utilization of large group donations, permits the storage of uncommon blood groups, and eliminates the problem of outdating of blood in the blood banks.

4. *Dosification of tranfusions.* The administration of frozen red cells should be accompanied by a better dosification of transfused red cells. So far, a transfusion is done utilizing the pint as the dosification unit, which is very inaccurate because one pint of blood has been found to fluctuate from 200-300 gm red cells. With a computation of the red cell mass in grams, blood replacement should be greatly improved and blood transfusion should become the accurate scientific procedure that it deserves to be.

5. *Oxygen transport.* Blood stored at refrigeration temperatures loses its oxygen transporting capibilities as much as three-fourths by the 21st day, as monitored by the concentration of 2-3 DPG.[6] The concentration of 2-3 DPG in frozen red cells however, remains stationary as of the day in which the freezing took place. Therefore, it has become customary to freeze cells not later than the fifth day to assure optimal hemoglobin function.

6. *Component therapy.* The use of frozen red cells allows for the availability of other blood components which have been separated from the whole blood at an early stage and processed into platelet packs, fresh frozen plasma, and other components. In a well established blood bank, these components should be available on a routine basis to match the increasing needs for platelet packs in oncologic therapy or fresh frozen plasma in coagulation therapy.

7. *Prevention of serum hepatitis.* Frozen red cells decrease the dangers of serum hepatitis.[7, 8] The risk of this disease has already been lessened by the pre-testing for HAA, but it still occurs in conventional transfusions. However, it is unknown in patients who have received frozen red cells. The explanation is not clear and it is speculated that the washing of the red cells removes the hepatitis associated antigen, as it removes surface attached antibodies and even bacteria.

8. *Emergency transfusions.* The possibility of the use of O negative blood as a universal donor is again true, since large amounts of this type of blood can now be stored and processed on short order in the event of an emergency.

Administrative Considerations

The advent of freezing techniques in a community blood bank or hospital must be supported by an adequate administrative scheme. The main limitations so far have been the high cost/unit of frozen red cells and the assignment of who will receive the high cost blood and the conventional blood. The most successful institutions have actually spread the cost of the frozen blood to all blood recipients, and dispense conventional packed cells and frozen packed cells without distinction.

The advent of autologous transfusion certainly may simplify the replacement policies prevalent in most blood banks, since the patient provides his own blood. An administrative charge is still necessary but this is also minimized in cases in which blood can only be used for the same donor and extensive testing is not necessary.

A frozen blood program may facilitite the upkeep of gigantic inventories extending to states or the nation as a whole, from which a large stock of unusual blood groups can be drawn. A national blood policy obviously will make use of these capabilities in time of emergency.

A well established service for frozen red cell transfusion is, so far, the only way for a stress-free blood bank operation.

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Clinical Notes

A Near-Painless Method of Sigmoidoscopy

LESLIE F. EATON, M.D., *Salina*

YEARS of experience from the perspective of observer, examiner, and examined have impressed on the author the anguish of the patient undergoing sigmoidoscopy. The multiple kinks of the retro-sigmoid colon are generally straightened for passage of the scope by inflation of a preceding air bag which inflicts excruciating pain on the patient. The following method has been devised to alleviate much of the pain of this procedure:

With the bowel adequately cleansed by whatever method is preferred, the patient is advised of the procedure to be followed. The patient is then placed on an examining table that preferably will allow 90-degree flexion at the hips with the patient in knee-chest position and weight bearing only on the knees and chest, keeping all weight off the abdomen and allowing enough space here for future use.

Place the headrest in position against the head, arranging for weight to be borne primarily by hands and arms as the head is tipped down to approximately a 45-degree angle.

With the patient in this position, use your hand to rather vigorously jiggle the abdomen, thus inducing the intestines to gravitate cephalad. In a high percentage of cases, if there are no restricting adhesions of pelvic masses, this will straighten the bowel past the rectal valves to the full length of the scope. Very little, if any, air is needed, and then not for inflation—a few gentle pulsations on the bag will suffice to determine if the scope is pointed in the proper direction for further advancement. On numerous occasions the scope has fallen the full length of 25 cm after it passed the valves.

The most revealing part of the examination is, of course, accomplished as the scope is withdrawn.

All the above is predicated upon the assumption that adequate lubrication—preferably an anesthetic gel—is utilized.

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Current COMMENT

A Diagnostic Approach to the Patient With a Chronic Diarrheal Disorder

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Ed. Note: This is the first in a series of articles prepared by the Department of Medicine at the University of Kansas Medical Center, covering the diagnosis and management of common clinical problems. Each of the subspecialty divisions within the Department of Medicine will contribute concise and readable essays appropriate for continuing education of primary care physicians. Each essay will be followed by several self-assessment questions with the answers given on a separate page of THE JOURNAL. A member of the faculty in the department will author or co-author each article.

Subjects to be covered in this and following issues include: "An Approach to Diarrhea," "Chronic Hepatitis," "Acute Bacterial Meningitis," "Nephrolithiasis," and "Evaluation of the Arthritis Patient." This series will provide the practicing physician with access to ongoing education programs at the University of Kansas Medical Center. Much of the material will be drawn from the Medicine House Staff Conferences as well as Medical Grand Rounds. Comments from readers of THE JOURNAL are invited.

Introduction

BOWEL HABITS of healthy persons vary widely. The term diarrhea may have little meaning unless there has been a change in the customary bowel patterns of an individual. Diarrhea can be defined as an increase in frequency, fluidity, or volume of bowel movements. In essence, diarrhea reflects malabsorption of water and electrolytes.

This review will cover four major areas: (1) the pathophysiologic basis of diarrheal disorders and the mechanisms by which diarrhea occurs; (2) a classification of chronic diarrheal disorders; (3) the irritable bowel syndrome, with special emphasis on criteria for establishment of the diagnosis and exclusion of other disorders which may simulate this condition; and (4) some practical considerations in the management of patients with diarrheal disorders.

Pathophysiology of Diarrheal Disorders

Physiology of fluid secretion and absorption by the gut. It is not generally recognized that very large volumes of fluid enter and leave the human intestinal tract each day. From *Table I,* it can be seen that approximately 2 liters of fluid from the diet, 1 liter of saliva, 2 liters of gastric juice, and 1 liter of small bowel secretions enter the proximal small bowel. Of the approximately 9 liters presented to the duodenum and jejunum, 4-5 liters are reabsorbed from the jejunum, 3-5 liters from the ileum, and 1-2 liters from the colon. As a result, stools normally contain 0.1-0.2 liters of fluid daily weighing 100-200 gm. For practical purposes, diarrhea can be defined as a 24-hour stool weight exceeding 300 gm on the average Western diet. The absorptive capacity of the colon is approximately double the amount of fluid which is delivered from the small bowel. Therefore, any disorder that results in more than 2 liters of fluid being presented to the colon can be expected to result in diarrhea. This could result from a variety of disorders such as: (1) infection with a toxin-producing micro-organism which causes intestinal secretion; (2) intestinal hypermotility as in hyperthyroidism; and (3) impaired absorption of fluid and electrolytes from the small bowel

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as in nontropical sprue and regional enteritis.

Mechanisms by which diarrhea occurs. It is not generally appreciated that diarrhea may result from several different types of abnormalities. Important mechanisms responsible for diarrhea are listed in *Table II.* First, diarrhea can occur because of alterations in the intestinal mucosa. Second, diarrhea may occur because of alterations in water and electrolyte transport without mucosa histopathologic lesions as, for example, after ileal resection and with certain tumors. Third, diarrhea may be on an osmotic basis; a prototype diarrhea here is milk intolerance due to lactase deficiency. Finally, diarrhea may be due to deranged intestinal motility and occurs with diverticular disease, hyperthyroidism, and laxative abuse

Classification, diagnosis, and management of chronic diarrheal disorders. Table III presents a classification of diarrheal disorders found to be clinically useful. In addition, *Table III* contains pertinent information on diagnosis and management of chronic diarrheal disorders. This classification can be remembered by utilizing the mnemonic shown at the bottom of *Table III.* An important cause of diarrhea is inadvertent dietary indiscretion. It is surprising how infrequently physicians take an accurate dietary history in patients with complaints of diarrhea. It is important to question the patient in some detail with regard to the intake of tea, coffee, cola beverages and simple sugars, as excessive ingestion of any of these nutrients can result in diarrhea.

TABLE I

APPROXIMATE VOLUMES OF FLUID ENTERING AND LEAVING THE HUMAN INTESTINAL TRACT DAILY*

Source	*Liters Into Lumen*	*Liters Resorbed From Gut*	*Fluid Presented to Gut*	*Efficiency %*
Diet	2	Jejunum	4-5/9	50
Saliva	1			
Gastric juice	2	Ileum	3-4/4-5	75
Bile	1			
Pancreatic juice	2	Colon	1-2/1-2	> 90
Small bowel	1			
Total	9			

Stools normally contain 0.1-0.2 liters of fluid daily weighing 100-200 gm. Accordingly, an operational definition of diarrhea is stool weight > 300 gm on the average Western diet.

* Modified from Phillips, S. F. Diarrhea—A broad perspective. *Viewpoints on Digestive Disease* 7:5, November 1975.

Infectious enteritis is an important cause of chronic diarrhea. The diagnosis of an infectious enteritis is usually established by demonstrating enteric pathogens in the stool. An important additional test, which can suggest the presence of infectious enteritis, is a Wright stain

TABLE II

MECHANISMS BY WHICH DIARRHEA OCCURS

I. *Alterations in the Intestinal Mucosa*
- A. Alterations in Permeability
  1. With damaged mucosa (nontropical sprue)
  2. Without damaged mucosa (bacterial toxins, *i.e.* toxigenic *E. coli*)
- B. Mucosal Injury with Histopathological Lesions
  1. Direct penetration with invasive organisms (Salmonella, Shigella)
  2. Inflammatory bowel disease (ulcerative colitis, regional enteritis)
- C. Drug-Induced Mucosal Injury (neomycin, colchicine, clindamycin)

II. *Alterations in Water and Electrolyte Transport Without Mucosal Histopathologic Lesions*
- A. Impaired Water and Electrolyte Transport
  1. ↑ passage of bile salts to colon → cathartic effects (ileal resection, ileal disease, after vagotomy and pyloroplasty)
  2. ↑ fecal excretion of hydroxy fatty acids → cathartic effects (ileal resection, steatorrhea, cathartics such as ricinoleic acid)
- B. Neoplasms with and without Elaboration of Secretagogues
  1. Zollinger-Ellison syndrome (gastrin)
  2. Carcinoid syndrome (serotonin, prostaglandins)
  3. Villous adenoma
  4. Pancreatic islet cell tumors (vasoactive intestinal peptide)

III. *Osmotic Diarrhea*
- A. Disaccharidase Deficiency Syndromes
  1. Primary lactase deficiency → milk intolerance
- B. Postsurgical
  1. Rapid gastric emptying (pyloroplasty, antrectomy, gastrojejunostomy)
  2. Intestinal resection
- C. Nonabsorbable Osmotically Active Agents
  1. Carbohydrates (lactulose, sorbitol, mannitol)
  2. Antacids
  3. Saline purgatives (phosphates, magnesium salts)

IV. *Deranged Intestinal Motility*
- A. Diverticular and Prediverticular Disease
- B. Hyperthyroidism
- C. Laxative Abuse
- D. Diabetes Mellitus (selected cases)

TABLE III

CLASSIFICATION, DIAGNOSIS AND MANAGEMENT OF CHRONIC DIARRHEAL DISORDERS

Cause	*Examples*	*Key Elements in Diagnosis*	*Treatment*
1. Iatrogenic dietary factors	Excess tea, coffee, cola beverages, simple sugars	Careful history taking	Appropriate dietary modifications
2. Infectious enteritis	Amebiasis Giardiasis	Demonstrate leukocytes in stool Identify trophozoites or cysts in stool and duodenal aspirate (giardiasis)	Amebiasis—metronidazole diodoquin antibiotics Giardiasis—metronidazole
3. Inflammatory bowel disease	Ulcerative colitis Regional enteritis	History: diarrhea, abdominal pain, rectal bleeding Sigmoidoscopy, barium enema, UGI and small bowel series	Sulfadiazine Corticosteroids
4. Irritable bowel syndrome	See *Table IV*	See *Table IV*	Dietary modifications Antispasmodics See text
5. Lactose intolerance	Milk intolerance	Milk → abdominal pain, diarrhea, gas, bloating. Cessation of milk drinking → amelioration of symptoms Lactose load (1 gm/kg) → exacerbation of symptoms and blood glucose fails to rise > 20 mg/100 ml	Discontinue milk
6. Laxative abuse		Add few drops of NaOH to stool. Because most laxatives contain phenolphthalein, the stool will turn red.	Discontinue laxatives
7. Drug-induced	Antacids, antibiotics (clindamycin, lincomycin, amphicillin, penicillin), colchicine, PAS, lactulose, sorbitol	Careful history taking and review of medication.	D/C offending drug
8. Diverticular and prediverticular disease		History: intermittent symptoms P.E.: Palpable LF. colon Barium enema: diverticulosis and/or muscle hypertrophy	High fiber diet. Avoid: corn, nuts, peanuts, kernel-containing foods.
9. Malabsorptive disease	Sprue Pancreatic insufficiency	UGI plus small bowel x-rays; tests of intestinal absorptive function: D-xylose, stool fat, Schilling test, serum carotenes, calcium, albumin, cholesterol, iron, prothrombin time	Appropriate for the underlying disorder
10. Metabolic	Diabetes mellitus Hyperthyroidism Adrenal insufficiency	Abnormal blood glucose levels ↑ T4, ↑ RAI uptake ↓ plasma cortisol, ↓ response to synthetic ACTH	Appropriate to the underlying disorder
11. Mechanical	Fecal impaction	Rectal exam	Remove impaction
12. Neoplastic	Carcinoma of the pancreas Carcinoid syndrome Villous adenoma Medullary carcinoma of the thyroid Tumors producing V.I.P. (vasoactive intestinal peptide) Gastrinoma	Suspect the diagnosis	Surgical

Mnemonic to remember the classification: I, I, I, I, L, L, D, D, M, M, M, N

of a stool specimen with a search for leukocytes. There is only a relatively small number of disorders that cause increased excretion of leukocytes in the stool. These disorders include shigellosis, salmonellosis, typhoid fever, infection with an invasive *E. coli,* amebiasis, and pseudomembranous enterocolitis. Other noninfectious disorders that can give rise to increased fecal excretion of leukocytes include drug-induced injury to the colonic mucosa and the inflammatory bowel diseases, ulcerative colitis, and regional enteritis. By contrast, viral enteritis, infection with toxin-producing *E. coli,* non-specific diarrheas, and most malabsorption syndromes are not associated with increased fecal excretion of leukocytes.

To recapitulate, an important screening test in evaluating the patient with a diarrheal disorder is to examine an appropriate stool specimen for leukocytes. If leukocytes are present, there are relatively few disorders that need to be considered.

Another infectious disorder that can give rise to chronic diarrhea is giardiasis. This is endemic in many parts of the world, but is also present in many parts of the United States including Colorado, the upper Midwest, and the southeastern United States. The diagnosis of giardiasis is suspected in an individual who has developed diarrhea that persists for more than two weeks and is associated with malodorous stools, modest weight loss of 5-10 lbs, minimally abnormal tests of intestinal aborptive function, and absence of other systemic symptoms. This diagnosis is established by identifying the trophozoite stage of the parasite in diarrheal stools, duodenal washings, or jejunal biopsies. A 10-day course of metronidazole is usually effective in eradicating the infection. Amebiasis is another common disorder that may be mistaken for infectious enteritis, ulcerative colitis, granulomatous colitis, and drug-induced colitis. The diagnosis can be established by demonstrating trophozoites in the stool. Alternatively, blood samples can be sent to the state laboratories or the Center for Disease Control in Atlanta, Georgia, for the indirect hemagglutination test for amebiasis, and this is positive in over 90 per cent of cases.

The inflammatory bowel disorders, ulcerative colitis, and regional enteritis are being observed with increasing frequency. A diagnosis of ulcerative colitis is usually established employing composite clinical criteria. These include: (1) a history of diarrhea, abdominal pain, rectal bleeding; (2) a compatible sigmoidoscopic examination; and (3) an abnormal barium enema examination. Similarly, the diagnosis of regional enteritis is usually suspected with the clinical findings of crampy abdominal pain, diarrhea, fever, palpable abdominal mass, and evidence of perirectal disease. A definitive diagnosis is made upon examination of biopsy specimens of the small or large bowel. Both sulfadiazine and corticosteroids have been shown to be effective in treating idiopathic ulcerative colitis and regional enteritis.

Lactose intolerance due to a deficiency of intestinal lactase is a common disorder affecting 6-10 per cent of the white population and 50-70 per cent of the black population The incidence is also quite high in certain ethnic groups, *i.e.,* individuals of Greek, Mediterranean, or Oriental extraction. The diagnosis is suspected from the history of milk giving rise to symptoms such as crampy abdominal pain, diarrhea, gas, and bloating. It is important to establish this diagnosis, since cessation of milk ingestion will lead to amelioration of symptoms. The diagnosis can be made by performing a lactose load of 1 gm/kg. A positive test is defined as one in which ingestion of lactose leads to: (a) exacerbation of symptoms; and (b) failure of blood glucose to rise at least 20 mg/100 ml in two hours.

Occult laxative abuse is a condition that must be considered in all patients who present with chronic unexplained diarrhea. The diagnosis can be established by adding a few drops of sodium hydroxide to a stool specimen. Because most laxatives contain phenolphthalein, addition of sodium hydroxide to the stool will result in the stool turning red. Further addition of hydrochloric acid will render the stool colorless. Other clues to the presence of occult laxative abuse include hypokalemia, hyposthenuria with urine specific gravity less than 1.015, and muscle weakness.

Drug-induced diarrhea is a common problem. The key elements in this diagnosis are a thorough history and a review of all medications which the patient may be taking. A partial listing of drugs that can give rise to chronic diarrhea includes antacids, antibiotics such as clindamycin, lincomycin, ampicillin and penicillin, colchicine, para-amino-salicylic acid, lactulose, sorbitol, and Phenformin. Perhaps the most common agents giving rise to diarrhea are antacids, and it is important to specifically inquire about antacid usage. Many patients regard antacids as over-the-counter preparations, and not as drugs or medications.

A frequent cause of chronic diarrhea is diverticular and prediverticular disease. The term prediverticular disease refers to the presence of hypertrophy in the circular muscle layer of the sigmoid and left colon. One current theory holds that prediverticular disease is common in Western countries because of low fiber ingestion. In essence, extensive prediverticular disease results in narrowing of the colonic lumen and low grade incomplete obstruction. In this setting, it is not surprising that patients develop diarrhea or alternating diarrhea and con-

stipation punctuated by bouts of abdominal pain. The diagnosis can be suspected from the history of intermittent symptoms. Physical examination frequently reveals a palpable sausage-like mass in the left-lower quadrant which is the descending colon or the sigmoid colon. Barium enema examination may reveal sawtoothing suggestive of muscle hypertrophy with or without associated diverticulosis. It is important to establish this diagnosis since there is preliminary evidence indicating that a high fiber diet will result in amelioration of symptoms and partial reversal of the lesion of muscle hypertrophy. In addition, patients should avoid corn, nuts, peanuts, and other kernel-containing foods.

Malabsorptive disorders characteristically give rise to diarrhea. Examples of two prototype malabsorptive disorders are sprue and pancreatic insufficiency. The diagnosis of a malabsorptive disorder is usually established on the basis of the history, physical findings, and tests of intestinal absorptive function. The latter include a stool fat examination (both qualitative and quantitative), D-xylose test, upper gastrointestinal and small bowel x-rays, Schilling test for B_{12} absorption, serum carotene, serum calcium, serum albumin, serum cholesterol, serum iron, and prothrombin time. The latter blood tests are indirect indicators suggesting that intestinal absorptive function may be abnormal. Such tests can also be used to follow the patient after initiation of specific therapy.

It is important to note that several metabolic disorders can give rise to diarrhea, which can be chronic. Such disorders include diabetes mellitus, hyperthyroidism, and adrenal insufficiency. Appropriate treatment of the underlying disorder usually results in amelioration of diarrhea.

An important cause of diarrhea, and one that is frequently not considered in a differential diagnosis of diarrhea, is incomplete mechanical obstruction of the bowel due to fecal impaction. This is usually diagnosed by rectal examination, and removal of the impaction usually results in cessation of diarrhea.

Finally, neoplastic disorders frequently give rise to diarrhea. It is seldom recognized that approximately 20 per cent of patients with carcinoma of the pancreas present with diarrhea and a clinical picture simulating the irritable bowel syndrome. Other neoplasms frequently associated with diarrhea include carcinoid syndrome, villous adenoma, medullary carcinoma of the thyroid and tumors producing vasoactive intestinal peptide, and the most common of these tumors are adenomas of the pancreas and bronchogenic carcinoma. The above classification of diarrhea disorders can be remembered by using the mnemonic I, I, I, I, L, L, D, D, M, M, M, and N.

Irritable Bowel Syndrome

General Considerations. Irritable bowel syndrome (IBS) is a very common problem. IBS is known by a number of synonyms including functional bowel disease, irritable colon, mucous colitis, and diarrhea of unknown cause. Criteria that have been found useful in establishing a diagnosis of irritable bowel syndrome are listed in *Table IV*. The usual criteria include the following: (1) symptoms of abdominal pain, diarrhea, or alternating diarrhea and constipation; (2) absence of systemic symptoms such as anorexia, weight loss, and fever (if these symptoms are present, another diagnosis should be suspected); (3) absence of hematochezia, melena, or occult blood in the stools (this underscores the need to do a rectal examination and search for occult blood in the stool); (4) normal sigmoidoscopic examination; and (5) normal barium enema examination. Additional criteria to be considered if symptoms persist include: (1) a normal stool weight of less than 300 gm/24 hrs; (2) absence of steatorrhea or other evidence of malabsorption; and (3) normal upper GI and small bowel series. In some cases, weighing of 24-hr stool collections may establish the need for more extensive study. This

TABLE IV

DIFFERENTIAL DIAGNOSIS OF IRRITABLE BOWEL SYNDROME (IBS)

A. *Criteria Useful in Establishing Diagnosis of IBS*
   1. Usual criteria
      a. Symptoms: abdominal pain, diarrhea, alternating diarrhea and constipation
      b. Absence of systemic symptoms: anorexia, weight loss, fever
      c. No hematochezia, melena, or occult blood in the stools
      d. Normal sigmoidoscopy
      e. Normal barium enema
   2. Additional criteria if symptoms persist
      a. No increased stool weight (24-hr stool weight $\leq$ 300 gm)
      b. No steatorrhea or evidence of malabsorption
      c. Normal upper GI and small bowel x-rays

B. *Differential Diagnosis of Irritable Bowel Syndrome*
   1. Iatrogenic dietary intolerance
   2. Milk intolerance
   3. Diverticular and prediverticular disease
   4. Drug-induced diarrhea
   5. Subclinical carbohydrate malabsorption
   6. Idiopathic bile acid malabsorption
   7. Irritable bowel syndrome not associated with an underlying disorder

is an important screening test.

In approaching the differential diagnosis of irritable bowel syndrome, a detailed history should be obtained and the physician should know the onset, duration, and organic qualities of the symptom complex (such as night stools and soiling). The patient should be asked to describe the appearance of the stool and to list all drugs taken. Examination of the stools should include tests for ova and parasites, blood, fat, pH, and a search for fecal leukocytes. The presence of steatorrhea underscores the need for additional tests of intestinal absorptive function.

In approaching the patient who has a symptom complex suggestive of irritable bowel syndrome, it is important to remember that several disorders may simulate the irritable bowel syndrome. These are also summarized in *Table IV*. Such disorders include iatrogenic dietary intolerance, milk intolerance, diverticular and prediverticular disease, and drug-induced diarrhea. Subclinical carbohydrate malabsorption is probably more common than is generally suspected. In this disorder, dietary carbohydrate is incompletely absorbed. Accordingly, if excessive amounts of carbohydrate are ingested, it will pass into the colon where it is acted upon by bacteria giving rise to products such as lactic acid, volatile fatty acids, and monosaccharides. This in turn can give rise to explosive diarrhea and crampy abdominal pain analogous to the patient with milk intolerance and lactase deficiency ingesting milk.

A relatively newly recognized disorder that mimics irritable bowel syndrome is idiopathic bile acid malabsorption. This disorder is due to a defect in the reabsorption of bile salts by the terminal ileum. As a consequence, bile salts pass into the colon where they exert their cathartic effect. A number of patients with irritable bowel symptoms for as long as 30 years have been studied and found to have incomplete absorption of bile salts. Cholestyramine, a bile salt sequestering agent, provides very effective treatment for this disorder. This effectiveness of cholestyramine in patients with irritable bowel syndrome might well be due to the fact that a certain subset of patients being treated with cholestyramine actually have idiopathic bile salt malabsorption.

After all of the above disorders are excluded, we are left with the patient who has idiopathic irritable bowel syndrome, *i.e.*, irritable bowel syndrome not associated with an underlying disorder. Such patients frequently have either painless watery diarrhea or crampy abdominal pain and diarrhea. They are awakened at night by crampy abdominal pain or diarrhea. There is an appreciable incidence of hypochondriasis, other psychosomatic complaints, subclinical depression, and other subclinical psychiatric abnormalities. Such patients often require frequent visits with a sympathetic physician who is patient and will listen to their complaints. In this setting, it is necessary to deal with the patient's bowel complaints and, after an appropriate workup, offer reassurance of help in management of the symptoms. Antispasmodic and anticholinergic drugs have been used in this setting with variable success.

Summary

There are several practical considerations in the workup and management of patients with diarrheal disorders. A careful dietary history should be obtained with special emphasis on coffee, tea, cola beverages, milk, milk products, and simple sugars. Similarly, a detailed drug history should be taken inquiring specifically about antacids, antibiotics, and laxatives. A history of anorexia, weight loss, or fever should suggest a diagnosis other than irritable bowel disease. A stool specimen should be examined for fecal leukocytes using either a Wright's or methylene blue stain. Stools should also be examined for ova and parasites and cultured for enteric pathogens. Rectal examination, sigmoidoscopy, examination of the stool for occult blood, and barium enema should be routine in all patients with chronic diarrhea. At this juncture, a tentative diagnosis can often be made and appropriate therapy initiated. If this is not the case, a 24-hour stool weight should be obtained as this is a useful additional screening test that can indicate the need for further studies. If the stool weight is increased, additional studies, such as an upper gastrointestinal and small bowel series, stool fat determination and other tests of intestinal absorptive function, should be obtained. If the patient has profuse watery diarrhea, or if there is other evidence to suggest a secretory diarrhea, a hormone-producing tumor, villous adenoma, and occult laxative abuse should be excluded.

Questions

1. What are the general mechanisms by which diarrhea occurs?
2. What are the criteria for establishing a diagnosis of irritable bowel syndrome?
3. What are the criteria for a diagnosis of lactose intolerance due to primary lactase deficiency?
4. If 0.1 NaOH is added to a stool specimen and it turns red, which of the following disorders should be diagnosed?
   a. Lactose malabsorption
   b. Regional enteritis
   c. Laxative abuse
   d. Ulcerative colitis

e. Sprue

5. In which of the following disorders is/are there increased fecal excretion of leukocytes?
   a. Shigella gastroenteritis
   b. Viral gastroenteritis
   c. "Turista" due to toxigenic *E. Coli*
   d. Irritable bowel syndrome

(Answers on page 267)

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A Comparison

(Continued from page 250)

tients who have positive RAST, but negative skin tests. For individual antigens, the rate of discordance was as high as 32 per cent. Since more than half of RAST results (66.8 per cent) were significant at 2+ or greater, and there was generally poor correlation with scratch tests, it appears that scratch tests are not sufficiently sensitive to be employed as the only diagnostic tool in the evaluation of allergic patients.

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Book Reviews

(Continued from page 242)

The next section (approximately 100 pages) is a potpourri of information on kitchen planning and storage, safety, appliances, clean-up, shopping, and nutrition. Two pages are devoted to metric conversion.

The next 80 pages are primarily devoted to simple, nutritious recipes, but even this section is liberally laced with helpful hints, many of them illustrated.

The final pages of the book include a tear-out page which can be mailed to the publisher with new ideas to share or requests for help in problem areas. It also includes a well-organized reference section listing sources of printed and filmed materials and the addresses of manufacturers of specialized tools and equipment.

The book itself is easily manageable—it is about $5\frac{1}{2} \times 8 \times \frac{3}{4}$ inches in size with a plasticized paper cover and loose-leaf spiral binding.—*E.B.*

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.

The President's Message

KaMPAC



The upcoming elections this year afford many opportunities for a change in the complexion and character of the Congress due to the retirement of many of its members. Senator James Pearson and Representative Joe Skubitz of Kansas are not seeking reelection.

Ms Sue Jenkins, Program officer of the Medicare Bureau; Health Care Financing Administration of HEW Region VII in Kansas City and with forty years of experience in the health care field, in a talk before the Medical Society of Sedgwick County spoke emphatically of the physicians' need to know their legislators and to express their views to them. "Your letters are read by them and are important to them. I know!" If we want to be heard by them and seek their support in medical matters, it is only right that they should be able to seek our support in their campaigns.

To think that they should listen to us because we're "right" or because we are important members of one of the learned professions is the height of naïveté. I am sorry to burst your bubble or throw sand on your ivory tower, but practical politics says, "Put your money where your mouth is." No one buys a Congressman with his money or support, but he does "buy," if you must, the right to have his viewpoints heard and his arguments considered. That is all anyone can ask or expect.

KaMPAC offers a method of effectively increasing the amount of financial support a candidate may receive from the medical community. KaMPAC has worked hard to become bipartisan and accomplish its objective of electing national and state legislators who, in general, agree with and support our philosophy. I believe it is accomplishing its mission and has been effective, and its support is appreciated and solicited by candidates. The most recent KaMPAC bulletin confirms this fact. I would urge all members of the Kansas Medical Society and their spouses to join KaMPAC. It is important this year as it is in every election year. The price of freedom is eternal vigilance and involvement!

Your individual support of your favorite candidate, both financially and personally, is also important and is encouraged. Many times this type of support helps to turn the tide in the community. The physician and spouse are leaders in their towns and cities and can do much to influence public thought and decision.

One often heard excuse is, "I can't afford to." We should be more interested in the politicians than they are in us! You know very well that they are not going to stop, and we can't either. Can you really "afford" *NOT* to be involved?

Let's get going and sign up NOW! KaMPAC has an ad in this issue. Read it on page 246 and then join. We need you now!

Fraternally yours,

Warren E. Mayer, M.D.

President



Editorial COMMENT

Laugh, Clone, Laugh

Doctor, we want to welcome you to our book discussion hour as we consider this new book, "In His Image"—I presume you are acquainted with it?

—Yes, as it happens, I have read it—which complicates things somewhat.

Complicates things?

—Yes. It's much easier to form an opinion about a book, especially a controversial one, if you don't read it. That's what I planned to do with this one but the publisher sent us a copy so I decided I might as well read it.

And now that you have read it?

—Well, the author, David Rorvik, has a certain amount of experience as a lay writer on scientific subjects. I hadn't read any of his earlier writings but that's neither here nor there. I would gather he has raised a few hackles in the scientific community in the past but again that's not necessarily to his discredit. But anyway, it was apparently his reputation as a writer acquainted with the personalities in the field of reproductive research that accounted for his being sought out as the catalyst in the controversial events he reports.

Which are?

—Briefly, he purports to record the first—reported, at least—successful cloning of a human being. If the book does nothing else, it will help this term on its way to becoming a household word. The writer, you see, is engaged by an unidentified man—self-made, unmarried, getting along in years, loaded with money—and obsessed with the idea of leaving behind a replica of himself. The writer's job is to bring together the necessary elements—human and technical—to do this little job. Now, this is no kindergarten stuff—siring a child with the help of some accommodating female, artificial insemination, or even propagation of an ovum fertilized by his sperm in a test tube. Such prosaic methods would introduce the female characteristics into the offspring and he wants the certainty not only that it will be male but that only his genetic material will be operating.

Sounds a little difficult.

—What it involves technically is obtaining and enucleating an ovum leaving only the cytoplasm, obtaining the free nucleus from one of his somatic cells, and fusing them into what would amount to a one-cell embryo. The nucleus he provides must of course be dedifferentiated so that it will not produce the parent tissue from which it came but be capable of—it is hoped—normal embryonic development. The autonomous cell must then be cultured to the morula stage and implanted in a receptive (in every sense of the word) female. From there on, we're back to the relatively simple process of pregnancy—although in this instance, this enceinte does seem to have received prenatal care like you never saw. Anyway, it's not, as you see, the garden variety science fair project. Boiled down, though, the contention is this: he says that's the way it happened and his critics say it couldn't have and it's the biggest hoax since Clifford Irving discovered Howard Hughes.

Do you think the book will be given much credence by the public?

—It has already contributed to talk about cloning and one of the criticisms has been that it will mislead people in their interpretations and expectations of legitimate genetic research. But I think there are two features which may influence the public attitude apart from the obvious ones. First, readers—and addicts of the tube—have long been exposed to a flood of science fiction themes. This pseudosophisticated public may not see it as the factual report the author claims it to be but I think rather as an interesting (and it is) but fictional exercise—possibly as a candidate for a prime time miniseries. Parental guidance advised, of course—meaning the parents will need the guidance.

And the second?

—The writer makes much of the fact that he does

not disclose the identities of the principals—the obsessed multimillionaire, the scientist who directs the team, the surrogate mother. Now if this had been accomplished in the traditional pattern of laboratory experimentation, this confidentiality would have been the appropriate course conforming to the legitimacy of the effort. But the fact is that it's produced as an expression of a rich man's selfish desire and we are introduced to it by way of the market place: the writer presents his case through commercial exploitation of the event. This in itself is no discredit to the literary effort—the narrative account is eminently more readable than the average scientific article—but he builds on a professed dedication to truth and morality to win the reader's support. The confidentiality of the one method becomes simply a lack of proof in the other and essentially what he says, since he can't name names or produce witnesses, is, "Gee, fellas, you gotta believe."

And do you?

—No—but then we can't cut this off so soon. I can't believe it happened as he says it did. But I don't think that matters—not to him certainly. What does matter is that the various capabilities exist and if it didn't happen this way this time, it will happen one day some way. The fact that it could happen takes less credence than the proposition that this particular group of participants brought it off under these particular circumstances. There is at least a certain satisfaction for traditionalists in noting that once the implantation was under way it still took—as the saying goes—nine months to have a baby. But we are told that we now have, growing up in California (where the strange is the norm and kids are used to confusion of parentage) a male child who is what? Son of only a father? Brother? The father himself in reissue?

But you say the writer is commercializing the event.

—That's evident in the very fact that he wrote the book. He makes a strong point of not having accepted any remuneration from the "father" as the go-between—a retention of his independence as he puts it—and publication of the book, even against the wishes of the "father," is viewed by the writer as a legitimate expression of his function as a medical reporter. Maybe this accounts for his international promotion of it. But, however profitable in book form, the significance of human cloning goes considerably beyond as sort of "Rover Boys on Mutation Walk." His style is one of apparent candor regarding the controversial aspects of the event and he seeks to validate the project with liberal use of references and quotations (which are not always germane or, in one instance at least, even accurate). But I found it to have a fictional aspect and became more engrossed in the characterizations than the process.

Such as?

—Well, this multimillionaire was motivated by an obsession to perpetuate himself (and only himself—no herself—though one can't help but hope the ribosomes in her cytoplasm will in some way outwit him). The scientist was motivated by *his* obsession to accomplish this "first" though he makes a great deal out of the accessory benefits of the work. And the writer, despite his soul-searching and occasional threats to withdraw, stays with it, writes about it, and promotes it as if he really hopes that in some way—not of his doing, of course—the cover would be blown. Not only would it help the book sales but how else can he finally get his story accepted as true?

And do you think that will happen?

—Remember, we're lapsing into the assumption that the story *is* true—which I don't accept as it stands. If it is true, he can't prove it without exposing the identities. If it isn't, he stands to be discredited. But the regrettable part of the disclosure would be the damaging effect of the relentless public probing of the two "innocent" participants—the surrogate mother and the child. You see, the scientist resorted to what he considered a legitimate subterfuge to gain her services. Once it was evident she was carrying (a good word in this instance) the child, she accepted her role but she was, quite bluntly, used. The child, of course, had nothing to say about it and if he exists we can only hope he can pursue a reasonably normal life of anonymity. But after all, we finally have what amounts to the interplay of three monumental egos—the man whose need for self-gratification was so great that he used, through his money, the scientist, the writer, this woman (as well as others who functioned in different phases of the project), and the child; the scientist who, however he rationalized it, let himself be used; and the writer who served, in his own portrayal, as the conscience of the effort but couldn't deny himself the gain of exposing it to the public.

You're not being overly kind.

—Kindness has nothing to do with it. Given the personalities and events presented in the book, I find it difficult to work up a great deal of respect for them, individually or collectively.

Then you're against cloning?

—What we're talking about, of course, is genetic engineering which I am in favor of—not, once more, that that makes any difference. The sheer force of intellectual curiosity will nullify the doubts, arguments, moratoria,

and so on which will play an appropriate but only delaying role in pacing the social progress toward its beneficial use. Our someday ability to correct genetic defects at their source is as logical and proper a use of our heads and hands as any aspect of medical care. Perhaps the production of individuals as described in this book will be accepted. This scientist preaches the desirability of such controlled and certain production over the haphazard matings of free choice, but I'm afraid the book will destroy its only possible good purpose—the promotion of thoughtful consideration of the legal and moral and ethical and social and political and—yes, medical—aspects of such activities. Not with hidebound prejudice or with the unbridled enthusiasm of amorality but, it would be hoped, with the intent of improving and enhancing the dignity and integrity of the individual—not his production and exploitation for willful and personal satisfactions. But I think the story was born—cloned if you will—of self-promotion, and I doubt if it will bring about the viable and healthy public examination the subject merits.

Thank you, Doctor, I see our time is up.

—Yes—and if the story *is* true, you may be quite right.—*D.E.G.*

Chronic Diarrheal Disorders

(Continued from page 263)

Answers

1. See *Table II.*
2. See *Table IV.*
3. (a) Ingestion of a lactose load (1 gm/kg) precipitates the development of abdominal cramps and diarrhea.*

(b) After a lactose load the blood glucose fails to rise 20 mg/100 ml above fasting levels.*

(c) There is no evidence of a primary malabsorptive disorder or other diseases known to result in lactase deficiency.*

(d) The level of lactase in jejunal biopsy specimens is decreased whereas the levels of other disaccharidases are normal.

(e) Biopsy specimens of the mucosa are normal upon examination by light and electron microscopy.

(f) Fecal pH is ≤ 6.0.

4. c.
5. a, d.

* These are the most useful criteria clinically; items d, e, and f are not regularly checked.

Council Meeting

Report of Meeting Held May 10, 1978

A meeting of the Council was held on Wednesday, May 10, 1978, immediately following the adjournment of the Second House of Delegates Session at the Downtown Ramada Inn, Topeka. Warren Earl Meyer, M.D., President, called the meeting to order.

Present were the following Officers and Councilors: Drs. Lewis G. Allen, Kansas City; Philip J. Antrim, Anthony; Kenneth M. Boese, Manhattan; Clair C. Conard, Dodge City; Jack R. Cooper, Shawnee Mission; Herbert Fransen, Newton; Phillip A. Godwin, Lawrence; Donald D. Goering, Salina; L. William Halling, Hays; Herman W. Hiesterman, Quinter; John D. Huff, Kansas City; Robert W. Hughes, Lawrence; Warren E. Meyer, Wichita; Robert F. Moore, Hoisington; George W. Pogson, Pittsburg; Lew W. Purinton, Wichita; Ivan E. Rhodes, Wichita; Alex C. Scott, Junction City; Marvin C. Snowbarger, Emporia; Millard C. Spencer, Topeka; William C. Swisher, Wichita; Max E. Teare, Garden City; William K. Walker, Sedan; Roger D. Warren, Hanover; Kermit G. Wedel, Minneapolis; Evan R. Williams, Dodge City; Emerson D. Yoder, Denton; John O. Yulich, Kansas City.

Also present were Jerry Slaughter, Gary Caruthers, and Val Braun.

Doctor Meyer introduced the newly elected Councilors as follows: Drs. Roger D. Warren, District 1; Lewis G. Allen, District 3; Kenneth M. Boese, District 5; Stephen J. Smith, District 8; Herbert D. Doubek, District 9; Max E. Teare, District 17.

Drs. David E. Gray, Topeka, and Donald R. Pierce, Topeka, were appointed to the Editorial Board for a three-year term.

The 1979 Nominating Committee was appointed as follows: John D. Huff, M.D., Chairman; Drs. F. Calvin Bigler, Richard L. Brownrigg, G. Rex Stone, and Emerson D. Yoder.

All action in connection with the House of Delegates' decisions was deferred to the next meeting of the Executive Committee.

(Continued on page 294)

Woman's Auxiliary

Open Letter to the Doctors of Kansas

The changing of the guard for the KMS Auxiliary has become a reality. Under the capable leadership of Jackie Burnett, another successful year has been completed. An increase in AMAERF contributions resulted in checks in the amount of $23,684.01 to KUMC, and $1,168.61 to the Wichita Branch of KUMC. Through the diligent efforts of our membership committee, the count at the close of convention was 1306 members. We need the moral and financial support of every physicians' spouse if we are to be an asset to the Kansas Medical Society. The new membership chairman is our new Second Vice-President, Mrs. Doris Lentz, Topeka. Her Co-Chairman and Member-at-Large Chairman will be Mrs. Diana Scamman, Topeka.

Heading the AMAERF Committee this year are Mmes. Peggy Grillot and Lucia Jenney, Wichita. Your support in the past has been most helpful and we urge the continuation of that support.

Legislation Chairman, Mrs. Betty Moore, Caney, will be assisted by Mrs. Katie Pyle, Topeka, Mrs. Phyllis Bigler, Garden City, and Mrs. Carolyn Gilhousen, Shawnee Mission. They will keep us informed and alert us when help is needed on legislative issues.

Our emphasis this year will be "Health Education for Children and Youth." It is the privilege of each President to choose an emphasis committee. The obvious choice for the mother of eight children and the wife of a pediatrician is CHILDREN. We believe that to improve the quality of life we must start at the beginning. To this end, this committee will be promoting the use of the Learning Center on Body Pollution for the 4th, 5th, and 6th grade students. Our traveling center is now available for use throughout the state. We are introducing a new program for teenagers—SCORE—Student Council Offers Responsible Education. This means involving the students in the program, and is a program which has been proven most successful. Educational material used in this program will come from the National Foundation-March of Dimes and will cover such subjects as nutrition, drugs, alcohol, smoking, etc. We believe that young people, given accurate information, will make responsible decisions concerning their own bodies. Heading this committee will be Mrs. Betty Barker, Council Grove, and her Co-Chairman, Mrs. Arlene Parman, Topeka. Regional chairmen have been appointed for some of the major committees, namely health projects, membership, legislation, and AMAERF.

A new committee this year will be headed by Mrs. Jackie Burnett, Halstead, as Liaison to the KMS Committee on the Impaired Physician. Co-Chairman of this Committee will be Mrs. Mary Ann Penn, Topeka.

There are other active committees, but space does not allow for a full report on each and every one of them. As physicians, if you see a need in your own community, please advise your own local auxiliary or contact us if you do not have an organized auxiliary. We stand ready to help you in any way you deem advisable.

It is an honor and a privilege to serve as President of the Kansas Medical Society Auxiliary. We are grateful for the encouragement, appreciation, and pledge of support voiced by Dr. James Lowman, Dean of the Kansas University School of Medicine, Dr. John D. Huff, Dr. Warren E. Meyer, and Mr. Jerry Slaughter. To be an effective Auxiliary, we need your comments, advice, and criticism.

Sincerely,
Jean Crouch
President
Kansas Medical Society Auxiliary

KMS 1978 Annual Meeting



↑
Bob Moore defends the honor of Elk County under Bill Swisher's quizzical eye.



↑
Lew Allen and George Penn promote Jack Cooper's display on "The Impaired Physician."

Reference Committee B ponders Clair Conard's resolutionary remarks.
↓



Don Goering rises to a point of order.
↓



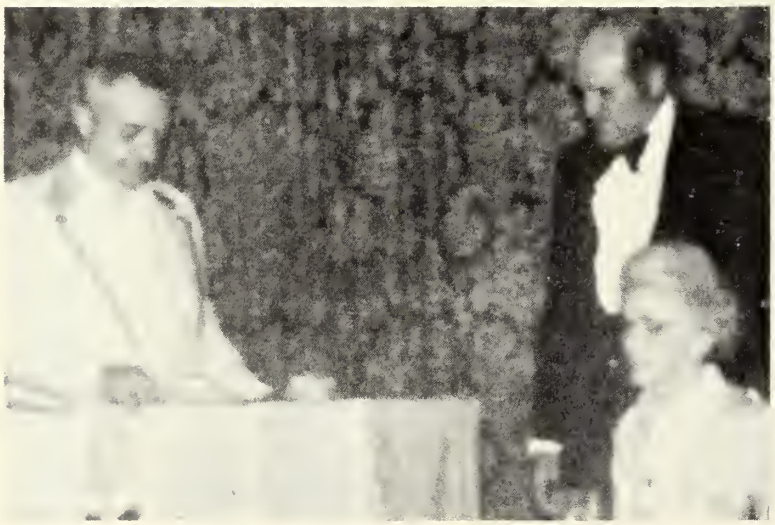
PRESIDENT'S BANQUET



The old order (forgive us, John) changeth, yielding place to new (you'll be sorry, Warren). ←

Jackie Burnett speaks for the distaff side. ↓





John Huff ponders a problem only a computer could solve—thanks to John Yulich and the Wyandotte County Medical Society. ←

SPORTS BANQUET



↑

Frank Bichlmeier demonstrates a single handed grip on his trophy to Bud Morris.



Ralph Hale enjoys the fruits of a good day's work.
←

Tom Meredith puts this year's claim on the Lattimore trophy.
↓





And Ken and Elizabeth Lohmeyer, Doris and Bill Lentz, and Royal and Betty Barker prove people can be civilized even after a long and busy day, enjoying the Dinner Theatre.



The Huffs and Bill Crouch made it up before breakfast—Early Birds.

KMS AUXILIARY







Edith Lessenden administers the Oath to Jean Crouch, President of the Auxiliary, and Kathy Wedel, President-Elect.

Jean gets her final prompting from Jackie Burnett—

—who joins Lela May Young in the happy relief of being "past."

Official Proceedings

1978 Annual Meeting of the House of Delegates

Transactions of the 119th annual session of the Kansas Medical Society are published in this issue of THE JOURNAL.

The resolutions are printed in numerical order under the Minutes of the Second House of Delegates meeting. Those resolutions which were not adopted but which were referred for further study or information are so indicated. The resolutions which failed to pass are retained in the official minutes at the Executive Office, but are not recorded here.

FIRST SESSION

The first session of the House of Delegates of the Kansas Medical Society met on Sunday, May 7, 1978, beginning at 9:00 AM, at the Downtown Ramada Inn, Topeka. The meeting was called to order by Clair C. Conard, M.D., Speaker, who explained the composition of the House.

The presence of a quorum was announced.

The Speaker announced the following appointments to the Reference Committees:

Reference Committee A: Alex Scott, M.D., Junction City, Chairman; Drs. Kenneth L. Derrington, Shawnee Mission; Vernon W. Filley, Pratt; Harry H. White, Topeka; R. Burnley White, Winfield.

Reference Committee B: Evan R. Williams, M.D., Dodge City, Chairman; Drs. Ernest P. Carreau, Wichita; Norman G. Marvin, Kansas City, KS; G. Rex Stone, Manhattan; James M. Stout, Hutchinson.

The following Tellers were appointed: Drs. John P. Brockhouse, Emporia; Louis M. Culp, Kansas City, KS; G. Rex Stone, Manhattan.

The Treasurer's Report was distributed to every member of the House of Delegates present. William K. Walker, M.D., Treasurer, solicited questions and comments. He announced that membership dues in the Kansas Medical Society are the third lowest in the nation.

The House heard the following reports, after which the session was adjourned at 12:00 PM.

Legislative Committee—
Jimmie A. Gleason, M.D., Chairman

It seems that every year the Legislative Committee works harder and harder. We physicians deal in absolutes, and we want everything that we can get; the legislative system being a compromise system, at best we have the opportunity to work through these things to develop a situation where we can get our point across, and that's the way we are doing it. We work very hard and we have always attempted to do this in an appropriate manner that is above-board.

Let's take a brief look at several bills of medical significance which were considered by the Legislature this year.

Medical School Tuition: A substantially amended version of SB-528, which provides scholarships (loans) to medical students in return for agreeing to practice medicine in Kansas has passed both Houses and been signed into law by the Governor. The new law does not increase tuition at the medical school (although tuition was raised to $3,000 per year by the Board of Regents earlier this year), but offers loans which pay tuition and other expenses in return for an agreement to practice medicine in Kansas upon completion of medical training. A list of underserved areas will be developed by the Department of Health and Environment with the assistance of the medical society and the medical school, and loan recipients can select prospective communities in which to practice from that list. If all the administrative details are completed, the loan program will undoubtedly be offered to this year's entering class.

Physicians Assistants: The controversial physicians assistants bill was finally passed by both Houses and signed into law by the Governor. The new law would require PA's to register with the Healing Arts Board, and it makes very clear that a physician employing a PA would "accept the ultimate responsibility for the actions of the PA under his or her direction and supervision." The section of the bill which would have authorized PA's to write prescriptions was deleted before final approval. The Kansas State Board of Healing Arts will be notifying all physicians of the new law and how it may affect the use of PA's in a medical practice.

Hospital Cost Containment: The Legislature considered two bills which would have established mandatory rate review commissions coupled with long-term planning for cost reduction in hospitals. Neither bill was passed by the Legislature, because it was felt that the efforts of the Kansas Hospital Association to voluntarily reduce costs, and the anticipated work-product of the State Health Cost Commission needed to be examined in greater detail before any restrictive legislation was

adopted. However, it is a certainty that legislation similar to that introduced this year will be re-introduced next year.

Brand Exchange: A much amended version of SB-108, the brand exchange bill, was finally approved by both Houses and signed into law by the Governor. Although the bill authorizes the substitution of generically-equivalent drugs, or "brand exchange," it is substantially less offensive than it was in its original form. The law is fairly liberal in the number of alternatives a physician has to authorize or not authorize brand exchange. A number of differing formats may be utilized for prescription blanks, or if the physician so desires, he may continue to use the blanks he has been using and make no change.

Laetrile: In the closing days of the session, the Legislature finally enacted the bill which legalizes the manufacture and distribution of laetrile in Kansas. The bill authorizes the administration of laetrile by physicians after patients have signed a "written informed request" which is essentially a formal disclaimer of the substance. Detailed record keeping would be required by physicians who administer laetrile and the Department of Health and Environment would be responsible for promulgating rules and regulations relating to the manufacture of the substance.

Nurse Practice Act: These amendments to the Nurse Practice Act basically amend the current law to recognize the expanded role nurse, or advanced registered nurse practitioner (ARNP). The definition of nursing was modified substantially, in effect, slightly liberalizing the scope of practice of advanced trained nurses. The new law also specifies certain criteria and educational requirements for nurses who want to be certified as ARNPs.

Chiropractors: None of the bills which would have expanded chiropractors' privileges were enacted into law by the Legislature. Senate Bill 90, which would have allowed chiropractors to withdraw blood, died in the House Public Health and Welfare Committee. Senate Bill 337, which would have authorized chiropractors to do school health certification exams, was killed by the House Education Committee.

Confidentiality of Medical Staff Committee Records: Although passed by the Senate, this bill which would make the records and proceedings of medical staff committees confidential and not subject to disclosure, was not passed by the House Judiciary Committee. The bill never came to a vote in the committee, and hence died for lack of action. At this time we are planning to reintroduce the bill, possibly with some amendments next year in cooperation with the Kansas Hospital Association.

Welfare Fraud and Abuse: Senate Bill 793 was enacted into law and would establish a state-funded telephone WATS line that any individual could use to report suspected cases of fraud and abuse. The bill was modeled after a similar law in the State of Washington where proponents claimed that it saved the state several hundred thousand dollars in potential fraudulent Medicaid abuses.

Temporary License Suspensions: The Kansas Medical Society introduced House Bill 3109, which would allow the Healing Arts Board to temporarily suspend a physician's license, without a prior hearing, if the Board determined that the physician's continuing in practice would be detrimental to patients' health. The bill was drafted by the KMS Committee on the Impaired Physician, and is considered a necessary provision as the Impaired Physician program is developing. It was modeled after a similar law in the State of Georgia, and although only used very rarely, it puts "teeth" into the enforcement of the Disabled Physicians Act by the Healing Arts Board.

Dispensing Physician: House Bill 2954 was a bill which would have required physicians who dispense drugs to register their offices with the State Board of Pharmacy. Although the bill passed both Houses, it was not finally enacted into law by the Legislature. The bill died in a conference committee because conferees could not agree on the Senate and House-passed versions of the bill.

In closing, I want to emphasize the importance of your cooperation; each of you, through the personal relationship that you establish with your own legislator, helps us to achieve significant effectiveness. There are, on both sides of the aisle, many legislators who are receptive to our viewpoints. In this election year, it is essential that you become acquainted with your candidate, so that you will be in a position to communicate your concerns during the next session.

This has been a good year; we appreciate your past support and solicit your continued assistance. The Legislative Committee will continue to work hard on your behalf. Thank you.

AMA Board of Trustees—Hubert A. Ritter, M.D.

I intend to discuss some of the problems confronting American medicine today, and what the AMA is doing to resolve—or mitigate—these problems. But first, let's take a brief look at the AMA itself, and at the need for membership support.

The usual approach is to talk about all the things that

the AMA has done for you lately, and then to urge every physician to join because we can do so much more together. Well, the AMA has done a lot for physicians lately, as I'll demonstrate a bit later on. Every single physician who values the future of medical professionalism and freedom of practice in this country should belong to the AMA. But let's take an entirely different approach. Let's accentuate the negative rather than the positive, because this too can be instructive.

If the AMA has public admirers, it also has public critics. If the AMA has medical supporters, it also has medical opponents. And if the AMA has friends in high places, it also has some prominent enemies—or at least people who talk like enemies on certain health care issues. All of these statements are true, of course; and it seems to me that they also are downright encouraging in some respects. Let me explain.

Since I am an officer of the AMA, you expect me to say that it is the only organization capable of effectively representing all physicians in this country, and that the AMA is medicine's rugged first line of defense against potentially damaging legislative and legal inroads. These statements are perfectly true. But you don't have to take my word for it. Take the word of our sometime adversaries instead. In any disagreement or dispute, those who hope to ultimately prevail must first discredit their strongest rivals. So it is that some of the more ambitious members of government reserve their loudest public lamentations for the AMA. So it is that some federal agencies, with their recent ill-advised forays into medical education and practice, reserve their best public shots for the AMA. And so it is that even certain splinter groups within medicine itself expend a great deal of their time and energy in attacking the AMA. In short, when such lengths are taken to discredit a particular institution, you can be sure that that institution is working extremely well. And given the seeming resentment in some government quarters against any opinions or proposals other than those of the chosen few, you can be equally sure that that institution is working on the right issues.

Certainly those of us at the AMA believe that we are working on the correct issues. For starters, we have eliminated the budgetary shortfalls of two years ago which seriously threatened the strength of the AMA. Our budget now is solidly in the black, our financial reserves are rejuvenated, and any operational fat has been pared away to bare bones and muscle. And heaven knows that we have dire need of money and muscle right now, because one of the largest AMA expense items today involves the legal fees associated with defending our profession from legal attack. There is the attempt by the Federal Trade Commission to undo the school-accreditation responsibility of the Liaison Committee on Medical Education jointly conducted by the AMA and the Association of American Medical Colleges. Seeing the AMA in every medical woodpile, the FTC claims that the Association has undue influence over the Liaison Committee, yet the FTC has offered no evidence that the committee lacks autonomy or embodies a conflict of interest.

While the AMA and the AAMC have helped guide the Liaison Committee in policy development, there is nothing in the policy that would limit the number of medical schools and physicians. As a matter of fact, since 1970 the United States physician population has increased four times as fast as the general population. Accredited U. S. medical schools have increased from 88 in 1966 to 116 today, with five more schools on the way. And the total medical school enrollment has jumped from 8,759 students in 1965-66 to 15,667 students in 1977-78—almost double.

The AMA also is mounting strong opposition to prevent misleading and deceptive patient solicitation under the guise of unrestricted advertising, another dangerous possibility posed by the FTC.

Last year, the AMA helped defeat a proposal in the Congress to extend FTC jurisdiction over non-profit organizations which would include state medical societies and even the Boy Scouts. Denial of that sanction means that the FTC will continue to be subject to the kind of legal challenge that the AMA is undertaking right now. There is no guarantee of success in the courthouse, of course.

On April 17, the United States Supreme Court affirmed without comment a lower court decision upholding the constitutionality of the 1974 Health Planning and Resources Development Act. But while the AMA—along with the State of North Carolina and its state medical society—may have lost that legal battle, we have by no means lost the war. On the one hand, our protracted legal battle has provided us with ample opportunity to make government—and the public at large—aware of the serious pitfalls in the act, and to propose appropriate measures, including legislative remedies. Thanks in no small measure to the AMA, for example, extension of certificate-of-need provisions to physicians' offices was deleted from a proposed bill in the U. S. House of Representatives.

On the other hand, the so-called "guidelines" for health planning, proposed last year by the Department of HEW, have suffered a disastrous setback in another court—the court of public opinion. To date, more than 70,000 letters of complaint have all but inundated

HEW's Washington offices, primarily because the guidelines ignore many of the realities of medical care, and because they could cause substantial disruption in the accessibility and provision of health services. Ironically, local health planning officials from communities across the nation were among the severest skeptics of the guidelines, especially with respect to the possible impairment or even closing of some local hospitals.

From the beginning of the planning act dispute, the AMA has been encouraging local physicians to get involved in—and thus help shape—the health planning structure; and to assist local physicians in that endeavor, we established the Ad Hoc Committee on Health Planning last December. This committee is now hard at work on three major projects:

1. AMA co-sponsorship of a series of regional health-planning workshops with the first one planned for September, and consideration of other ways to strengthen communications between state and county medical societies and local planning agencies;

2. The joint development with the Ad Hoc Committee on PSRO of model HSA/PSRO agreements which are currently being mailed to state and county medical societies, local PSROs, and specialty groups; and

3. The completion within a few months of an informational handbook covering health planning from the act itself to selected strategies for physician involvement.

During 1977, the AMA helped defeat, stall, or blunt several other major Congressional moves against medicine. Defeated were proposals to include laboratories in physicians' offices in new federal regulations covering clinical labs and additional proposals to subject physicians' offices to certificate-of-need programs, as recommended in several versions of the hospital "cost containment" proposals. Furthermore, while the AMA generally supported the principles behind the Medicare-Medicaid anti-fraud and abuse amendments signed into law by President Carter, we helped achieve some important modifications, including the following:

1. The definition of "shared health facilities"—originally encompassing almost all group practices—is narrowed more closely to the originally-intended "Medicaid Mills" and the like, thus generally exempting most practitioners and medical groups from cumbersome federal reporting and disclosure requirements;

2. Any Medicare-Medicaid review programs duplicating those of PSROs are waived, provided the latter are found to be performing their duties effectively. Also, authority for "program review teams" to evaluate medical care—thus duplicating PSRO review—is repealed; and

3. The period for a PSRO's conditional status, as well as the starting time for ambulatory care review under PSRO, are extended.

Late in 1977, the AMA added its strength to the successful drive to curb the federal imposition of foreign medical student "quotas" in our medical schools. Just a few weeks ago, the AMA strongly protested the public release of a report from the federal Council on Wage and Price Stability, a report which contains exaggerated estimates of physician income levels; and the familiar—but no less erroneous—assumption that our federation has practiced professional birth control for economic gain. In letters to President Carter and Council Chairman Charles L. Schultze, AMA Executive Vice President Jim Sammons pointed out that the report is replete with statistical and interpretive inaccuracies, and that many of the charges made are entirely without documentation.

Meanwhile, a detailed AMA analysis of the Council's report cites an article in the May 1977 issue of *Harper's* Magazine authored by Mr. Schultze. In that article, he emphasizes that in responding to current social issues, there is a tendency for those in government to use suspect tactics in justifying their programs and positions. Two of these tactics are to reduce the principals involved to the level of heroes versus villains, and to repeatedly stress the urgency of every problem. Unfortunately, by authorizing release of the Council report, Mr. Schultze appears to be practicing what he proscribes.

In any case, the record shows that the AMA is effectively representing practicing physicians in the courthouse and on Capitol Hill. And let me hasten to add that we have initiated or supported a wide range of positive programs—including legislative programs—in health care. Within the past few years we have initiated or supported programs—in some cases government programs—to upgrade care for the emotionally ill, the jail inmate, the American Indian, the migrant worker, the rural dweller, and the inner-city resident. And we have attempted to initiate or stimulate the development of answers for our society's major health care problems.

The rising cost of care is without doubt the most visible problem today. And like most physicians, we at the AMA are deeply concerned about costs as demonstrated by our creation of the National Commission on the Cost of Medical Care. As you know, the commission has made some sweeping recommendations for relieving problems resulting from escalating costs, recommendations which will be considered by AMA Delegates at our Annual Meeting in St. Louis this June. The commission itself was free-standing, with 27 representatives from various public and private institutions in our society.

As might be expected, the commission's 48 major recommendations have generated some disagreement, even among the commissioners themselves. But the commission recognized from the outset that if our society is to effectively resolve or alleviate cost problems, it will require a great deal of cooperation and some accommodation from everyone involved. And if the commission's recommendations have been controversial to some extent, their credibility has been affirmed in high places.

For example, U. S. Representative Dan Rostenkowski is one of the two most powerful health legislators in Congress, and as a result of the commission's recommendations—published last December—he has proposed that the private sector be given a last chance to get its cost-containment act together. This opportunity, in turn, is why the AMA has joined the American Hospital Association and the Federation of American Hospitals to do just that. We have formed a national steering committee on costs which has formulated its own recommendations for moderating the costs of care at the local level. Two primary goals are to reduce annual increases in hospital expenditures by two percentage points in 1978 and 1979, and to impose a moratorium on building of new beds. Every state now has its own steering committee working hard to prove that in seeking to moderate costs, as in other areas, the private way is the better way.

In closing these remarks, I could cite some other recent developments of more direct benefit to physicians, such as our continuing emphasis on scientific programming, including expanded continuing medical education opportunities; the continued establishment and evaluation of educational standards; and the extension of direct representation in our House of Delegates to medical schools and specialty societies.

This is not to say that the AMA is doing a perfect job or that everyone of its programs or policies will be agreeable to every physician. Heaven knows that I myself don't agree with everything the AMA does or says. But I do believe in the democratic way of doing things which includes consensus by majority vote, such as in the AMA House of Delegates. I also believe that on balance, the AMA is one of the most effective associations of its kind anywhere. And the average American believes that too, at least according to a pair of public opinion polls last year. Polls by Gallup and Market Opinion Research revealed that 71 per cent of the public has either a "great deal" or a "fair amount" of confidence in the AMA, and that physicians still rank first in public esteem in terms of honesty and ethics—slightly ahead of engineers and college professors, and considerably ahead of journalists, lawyers, and legislators.

It's just too bad that more physicians don't understand that the AMA works and that it's working on the right things. Because it won't be "certain well-meaning but medically-misguided people" in government and elsewhere who will ever seriously compromise the AMA and the best of American medicine. This will happen only if we ourselves let it happen. It will happen only if too many physicians sit around and complain rather than contribute time, effort, and money. It will happen only if too many physicians are unwilling to invest several hundred dollars per year to help preserve the quality of the medical schools from which they graduated, the specialties that they are certified in, and the magnificent profession that they are privileged to serve.

Well, these things won't happen. Because it's up to you and me and other physicians like us to see that it doesn't happen. It's up to us to make non-members understand that only an AMA strong in membership and united in purpose can assure the future of American health care. So let's get on with it.

Mediserve, Inc.—
Emerson D. Yoder, M.D., Chairman

Mediserve is an organization formed approximately two years ago jointly by the Kansas Medical Society and the Kansas Farm Bureau. It serves to help to obtain medical care for the underserved areas of Kansas, primarily in rural areas. The organization is operated by a board composed of representatives from the Kansas Farm Bureau and the Kansas Medical Society, three from each organization. The KMS members on this board have been Dr. Kenneth Graham, Dr. Don Goering, and myself.

The organization has now completed its first full year in operation, and it really is just getting off the ground. It functions by: (1) assisting medical students to gain admission to medical school by making recommendations to the admissions committee of the school; and (2) by offering limited financial support, if needed, to these students as they go through their phases at any level of their medical education, including residency.

The students or residents are asked to sign a contract to agree to practice a maximum of five years in an area of Kansas which has been declared an underserved area or a needy area in the field of primary care (this includes the fields of family practice, pediatrics, internal medicine, and obstetrics/gynecology). The students choose where they would like to practice, and Mediserve must approve or disapprove the location. We have the staunch support of the undertaking by the Kansas University from Dr. Dykes, and have been assured 20 places

under this plan. This year, the first year of functional activity, we had two days of interviews, one in Manhattan and one following that in Topeka, early in December. At that time we spoke with 30 students and their parents. Twenty-nine of these paid their admissions fee at KU. Of the 29, 14 were admitted to the regular screening process; 2 were approved, 2 were eliminated in the early selection process; 4 or 5 were placed on the alternate list. So, we are a little short of the quota that Dr. Dykes promised us and there is some re-negotiating taking place there. It is possible that some of these alternates may still be admitted.

One of the recent legislative activities by the Board of Regents and the Kansas Legislature, referred to by Jimmie Gleason, may either remove or alleviate the need for the loan portion of our organization. I say it *may*, because it is too early to tell whether we will still have students who would like to apply to us for loans, or whether they are all going to be on state scholarship as suggested by our new legislative activity.

The second aspect of this program—assistance in gaining admission to medical school—is still as important as before, because we had some students admitted this year who had tried before and were rejected. After we interviewed them and were convinced that they would make good physicians for active practice in the locations we are talking about, and made our recommendations, they were accepted by the medical school.

As we were being organized, our parent organizations —the Medical Society and the Farm Bureau—each agreed to contribute $10,000 a year to provide the funds for the student loans. These were not to be given as grants or contributions; these were to be made as loans repayable at a low rate of interest in each case.

Now this year there is going to be uncertainty on the dollar situation regarding the medical school tuition and their need for the loans. The board members representing each of the two organizations suggested that the sponsoring organizations keep this $10,000 pledge, while not actually making the current contribution until such time as it becomes clear that the funds will be used.

KaMPAC Board of Directors— Antonio Huaman, M.D., Chairman

During 1977-1978, the Board of Directors of the KaMPAC has taken the subject of medical politics to the grass-roots of the memberships of the Kansas Medical Society and the Auxiliary. We have done so whereever and whenever we have been invited as speakers. Our presentations dealt with the historic role physicians have played in the development of the United States as a republic, the role of physicians in the State of Kansas, and the necessity of practicing medical politics as part of our medical practices. We have discussed the Federal Election Act and the Federal Election Commission that regulate the political action committees, and the relationships between political action committees and membership organizations such as ours. We have been, in sum, introducing medical politics into the everyday practice of medicine because we believe that the social moment of our country calls for physicians to represent themselves in the public opinion and legislative process in order to preserve the high quality of American medicine.

During 1978, an election year, we have an extensive schedule ahead of us. Two members of the Kansas Medical Society, Drs. Ralph R. Reed and William R. Roy, are seeking the Senate seat being vacated by Senator Jim Pearson. We should support these candidates according to our political preferences and with the ultimate aim of having a Kansas physician representing Kansas and the American medical professions in the highest legislative body of our country. There are several personalities who are seeking a seat in the United States House of Representatives. Several of these personalities have supported our position in the Kansas Legislature, and I believe we should support them in their quest for a higher office. The Kansas Legislature will also be renewed this coming November, and we should be prepared to support friends of medicine in our own state legislature. The state laws affect our practices as much as the federal laws. Our only recourse to influence the unavoidable laws that will govern the practice of medicine is the access to state and federal legislators who could be receptive to our viewpoints.

This year, we will also step up our drive for membership. Your financial contribution to the political fund is essential to our political clout. Therefore, you may expect letters and telephone calls from members of the Board of Directors encouraging such contributions. To facilitate our membership drive, we have drafted a resolution for joint billing for KaMPAC and the county medical societies, for which I request the support of this House.

The financial support to candidates will be awarded according to a procedure just established by the Board of Directors. It consists of an interview of the candidate by a physician, filling out an evaluation form, and then discussion of his philosophy and possibilities of success before the Board of Directors. The decision to support a candidate is made by simple majority vote. We believe that, as custodians of your contributions, we should give them to the candidates with better possi-

bilities of success.

Before I finish my report, I will ask you all to join me in honoring the component societies which have contributed the greatest number of members to the Political Action Committee. We will be honoring the representatives of the Sedgwick County Medical Society for the largest number of KaMPAC members, and the Shawnee County Medical Auxiliary for the largest number of women KaMPAC members.

Finally, it is my pleasure to invite you all to the hospitality suite hosted by the Board of Directors of KaMPAC in the Governor's Club of this hotel at 4:00 PM today.

KaMPAC awards were received by Dr. Richard Cummings, Medical Society of Sedgwick County; Dr. Roger Warren, representing the Northeast Medical Society; and Mrs. Carol Loeffler, representing the Sedgwick County Medical Auxiliary.

KMS Impaired Physician Program—George M. Penn, M.D., Co-Chairman

Resolution No. 77-7 directed that the Society undertake a study for the possible solution of problems of physicians discipline in Kansas. An active committee, called the Committee for the Impaired Physician Program, is indeed now operational for dealing with the problem of impaired colleagues. We hope we will be able to identify, to compassionately deal with, and to rehabilitate those of us who are impaired.

I think it is time that we begin to recognize that we ourselves have needs, and that if "you will not help me" and "I will not help you," it is unlikely that we are going to get help.

A very significant piece of legislation, HB 3109, was passed this spring. It was a bit controversial, but with Jerry Slaughter's expert lobbying efforts, we now have the support of the law to help us in persuading colleagues to seek treatment when it is indicated. Hopefully this law will never have to be used, but it does provide the State Board of Healing Arts with the power to temporarily suspend, revoke, or limit the license without a hearing if there is evidence that continued practice is immediately harmful to the welfare of patients. This law, I think, is well worth your reading.

The next major action is to develop a Physician Contact/Support Committee, which is going to be composed of a number of teams of physicians who will be volunteering their time to be responsive to calls out in the state, meeting with physicians who have been determined to be impaired. That process is spelled out in the Statement of Purpose, following Resolution No. 78-16.

Crucial to any effort in identification and case finding, as well as in education and prevention, is the role of the physician's spouse. The Committee for the Impaired Physician includes representatives from the Kansas Auxiliary, who are serving as liaison from our committee to the Auxiliary.

Last January, a Conference on the Impaired Physician was held in Topeka. The interest and attendance were very encouraging to those of us who have worked on this project. Hopefully, a program somewhat similar but aimed at other conditions—neurologic and medical impairments—may be in the offing next January.

The Committee of the Physician Contact/Support teams will require, among other things, the learning of various techniques and skills of confrontation. There are plans in the next several months to have a workshop to that effect, that deals with those phases. It will be a workshop for physicians who are interested in the signs and indications of impairments and would like to learn more about identifying and confronting such physicians.

Another secondary but equally important aspect of this entire program is the remedial program to be directed at educating medical students, residents, and spouses. We have been contacted by KU and Wichita wives' groups interested in programs on substance abuse, marriage, and impairment.

Additionally, there will be a special issue of the JOURNAL OF THE KANSAS MEDICAL SOCIETY this fall that will be devoted entirely to the problem of impaired physician performance.

KMS Auxiliary—Jackie (Mrs. A. Dean) Burnett, President

The purpose of the Auxiliary is to assist the Kansas Medical Society in its programs to improve the quality of life for all people, promote health education, coordinate and advise the activities of county auxiliaries, and to support health-related charitable endeavors. The phrase "to cultivate friendly relations and promote understanding among physicians' families" will be deleted from Auxiliary Bylaws, as the IRS has determined that it is a self-serving purpose. It remains our conviction, however, that good relationships between physicians' families do serve the public's best interest.

Our year of "Commitment to Family and Community" has realized more new community projects by county auxiliaries than ever before. Greater personal involvement in their communities has indeed improved the quality of life through a wide variety of projects ranging from nutrition puppet shows for primary children to birthday parties and activities for the aged. County-

wide CPR training for the public, babysitting clinics, purchase of a resuscitator for a hospital, monitor for a sports arena, a conference on child abuse for the public, hygiene kits for day care centers and a learning center on body pollution for fifth- and sixth-graders are a few of the endeavors of auxiliaries to meet needs in their communities. To finance their projects, they have held flea markets, art auctions, book sales, garage sales, and bazaars. Many of these projects and fund raising events have been so innovative that the media have given extensive coverage. Such remarks as "I didn't know doctor's wives did things like this," or "Thank you. Will you do it again?" tell us that *what* we *do* makes a difference, both in the condition of our communities and the public concept of the medical family.

The Auxiliary joined the Kansas Medical Society in sponsoring a Conference on the Impaired Physician. It was an effort to inform and motivate physicians and spouses to be concerned about the plight of the impaired physician. It was a sensitive and positive effort to help the medical family.

A Learning Center on Body Pollution originally developed by the Wyandotte County Auxiliary has been duplicated by the state Auxiliary. It will be a traveling unit to be used in fifth- and sixth-grade classrooms over the state, consisting of a colorful and well developed variety of 12 games which provide facts about alcohol, tobacco, nutrition, and drugs. Hopefully, information gained will better equip children to make good decisions about their own bodies.

How can your Auxiliary better serve its parent organization? *By direction from you.* Joint efforts by county societies and auxiliaries can provide volunteer services to communities as well as reflect a more characteristic image of the profession.

For the first time, the AMA asked its Auxiliary to assist with a project—The Immunization Awareness Campaign. You never saw such a campaign! The results were successful and significant. Besides children getting their vaccines and parents learning why vaccines are still needed, physicians learned that they have a super support team in their Auxiliary.

My suggestion is that county physicians can utilize the skills of their spouses in suggesting a joint project, helping plan it, and then turning over the leg work to their greatest supporters.

This year it has been my pleasure to attend the KMS Executive Committee, Council, Legislative, and KaMPAC committee meetings. I am representing the Auxiliary on the Committee for the Impaired Physician, and I am looking forward to serving in education and prevention. One of my greater challenges has been to write a column in THE JOURNAL. It has been an honor to serve the Auxiliary and Society this year. The respect that I have always had for your profession has only deepened as I have come to know more physicians. Keep those ideals polished, your commitment regenerated, and may God bless each of you. You have more friends than you know. Thank you.

Constitutional Secretary—
Jack R. Cooper, M.D.

This is the Membership Report of the Kansas Medical Society for 1978:

Dues Paid Members	2,054
Resident Members	17
Emeritus Members	74
Leave-of-Absence Members	21
Personal Exemption Members	3
Retired Members	147
Service Members (Armed Forces)	3
Student Members	10
Honorary Members	3
Delinquent Members	295
Total	2,627

This compares with the membership in previous years as follows:

1970 Total Membership	1,921
1971 Total Membership	1,990
1972 Total Membership	2,003
1973 Total Membership	2,033
1974 Total Membership	2,078
1975 Total Membership	2,137
1976 Total Membership	2,205
1977 Total Membership	2,267

Necrology Committee—
David E. Gray, M.D., Chairman

It is reported that Gertrude Stein, as she lay dying, was heard to ask, "But what is the answer?" After a pause, she said, "But then, what is the question?" For those of us still struggling with the question, this is the time to recall our colleagues who, in the past year, have found the answer.

Name and Address	*Age*	*Date*
William W. Abrams, *Kansas City*	70	Oct. 20, 1977
Kenneth E. Conklin, *Abilene*	74	Sept. 22, 1977
J. O. Gilliland, *Herington*	68	Aug. 29, 1977
Leland L. Holbert, *Kansas City*	62	Nov. 10, 1977
Cecil C. Hunnicutt, *Sabetha*	73	Nov. 24, 1977
Victor R. Moorman, *Hutchinson*	61	Sept. 8, 1977
William J. Porter, *Pratt*	51	Aug. 17, 1977
Harcharan S. Sehdev, *Topeka*	41	July 7, 1977
Margaret L. Shiney, *Topeka*	50	Oct. 2, 1977
Francis M. Stone, *Fort Scott*	62	Oct. 29, 1977
Alonzo A. Towner, *Varadale, WA*	67	Sept. 23, 1977

Editorial Board—
David E. Gray, M.D., Chairman

Over the past few years, the reports of your Editorial Board have been marked by two characteristics: first, a doleful recitation of financial woes, and second, a relative brevity—relative, that is, to the editor's usual verbal flights as well as to some of the other reports. In the last two years, your largesse has spared you the former but you are still at my mercy for the latter.

In spite of the continuing erosion of inflation and the resulting cheerful candor with which our publisher advises us at frequent intervals that it's time to pass along another increase in cost, our financial position continues comfortable if not robust. In general, then, it has been a satisfactory year except for a brief period of poverty of a different sort. Our contributors apparently became so preoccupied with the threats of malpractice, satisfying audit committees, filling out sundry forms, counting up CME hours, and perhaps even practicing medicine that they forgot to create some of those gems of medical lore for which we were waiting. I am happy to report that our heart-rending appeal—or something—produced a response which saved THE JOURNAL from surpassing these reports in brevity.

Perhaps this literary drought was my punishment for making my annual invitations for your contributions somewhat perfunctory. Perhaps some of the mystique has been lost from our relationship as each of us has taken the other for granted. If this sounds like a build-up for a proposition for open marriage, forget it. Let this serve as a fervent renewal of that appeal and accept the fact that you're hooked up with a nag so you might as well start producing.

The successful production of THE JOURNAL during the past year can be accounted for in large part by the presence of Eleanor Bell who, as editorial assistant, has been of great help to us and has relieved our indefatigable managing editor of much of her burden. A good foundation in journalism, a warm interest in the welfare of the JOURNAL, and an engaging personality have made Eleanor a welcome addition to our group.

Veterans of these sessions know that no dedication to brevity will permit me to fail in the most important feature of these reports—the annual attempt to express our gratitude for that workaholic, Val Braun. The Board's and my personal admiration for her continues to grow and this is the only place I can make it a matter of record without fear of her deleting pencil.

And the concluding portion of my act continues to be the Board's annual expression of conceit. It is our pleasure to present to our president, John Huff, a bound copy of the Board's answer to *Hustler*.

The Kansas Foundation for Medical Care, Inc.—
L. William Halling, M.D., Acting President

In February 1972, a group of physicians, some of whom are on this podium, some of whom are in this audience, met to incorporate an organization they called The Kansas Foundation for Medical Care. It had several purposes. Perhaps the most important was to study and promote the improved methods and facilities for the delivery of health care, and to serve as the mechanism to provide members a means to contract terms and negotiate contracts with third-party purchasers of medical care. That was in February of 1972.

In October 1972, Congress passed the PSRO law, and Dr. Frank Collins, who had been mainly instrumental in the Foundation's creation, set about the task of formulating a working mechanism for implementing the national mandate of the PSRO law.

For approximately five or six years, the Foundation has struggled to retain its status as a planning PSRO, while the bureaucrats have concerned themselves with nitpicking about certain items of the KFMC by-laws. Last year, Dr. Kenneth Graham was President of the Foundation and, as you know, his health required that he diminish some of his duties. I was asked by the Board of Directors to assume the duties of the President on an acting basis until new officers were elected.

Dr. Collins, fortunately, has provided for us a beautiful staff, including Jim Agin as Executive Director, and more recently Larry Pitman as Associate Executive Director, and this staff has accomplished a great deal over the past months. We are now a "conditional" PSRO, although we are not, as I recall, considered "operational" for another four years. The Foundation is a creation of the Kansas Medical Society and, as such, will continue to make every effort to cooperate and work with the Kansas Medical Society.

This year the KFMC-PSRO has as its goal the delegation of PSRO review to Kansas hospitals. So far, 105 delegation applications have been received; by December of this year 75 hospitals will be delegated. Already six workshops have been held throughout the state and an additional four workshops are planned prior to the end of this year. These workshops will provide the information required by the hospital personnel to conduct the PSRO function in their hospital. Recently the list of reasons why hospitals should be delegated was developed by Dr. Collins, and distributed to the Chief of Staff of each hospital in Kansas. Those reasons are the basis for the Foundation and are:

1. To maintain local determination and control of utilization, MCE, and quality supervision;

2. To assure that the KFMC peers are carrying out the mandate of the PSRO law;

3. To preserve local control of quality assessment;

4. To demonstrate that physicians can provide leadership in quality evaluation and MCE organization without government intervention;

5. To demonstrate the commitment of physicians to the private enterprise system;

6. To assume the responsibility to try to control our destiny;

7. To provide our patients assurance of professional determination of their medical services;

8. To be involved in the entire aspect of medical economics as well as medical care; and

9. To be able to demonstrate that PSRO is legally and fundamentally a quality assurance program and not just a cost containment mechanism.

This then is our intent, and if we see that the direction is otherwise, we are prepared to stand on the basis of the original intent of the law, which speaks to the development of a quality assurance program.

Most of you believe that a gazelle is a fleet-footed African animal, but I think probably Gessell will be a name that you will be familiar with very shortly. Judge-Gessell has recently ruled, in response to a suit brought by a Ralph Nader group against the National Capital Medical Foundation, a single foundation representing the Washington, D.C. area, that the PSRO is indeed not subject to the confidentiality provisions of the PSRO rules and regulations, and that the Freedom of Information Act pertains to PSROs. The ruling was that PSROs actually are subdivisions of government. National PSRO organizations, as well as the National Capital Medical PSRO, will of course appeal this to a higher court. This will not at present affect any PSRO other than the National Capital Medical PSRO.

The implications of his ruling may very well sound the death knell to PSROs, unless Congress intervenes with appropriate confidentiality legislation. This will be one of the items that will concern you and us in the coming year.

We did elect a new board that we believe is representative geographically of all Kansas, and today we also elected a new slate of officers. The new slate of officers includes Dr. Lewis M. Culp (Kansas City), who is the new president, Dr. Richard M. Glover (Newton), myself, and Dr. George R. Learned (Lawrence), who will fill the offices of vice-president, secretary and treasurer.

Thank you.

The President—
John D. Huff, M.D.

My year of presidency has passed faster than water through an irritated colon. It has been a most enjoyable tour of duty. The opportunity to serve the most friendly, dedicated health care providers in the world was deeply appreciated. I should like to thank all those individuals who contributed to keeping the Kansas Medical Society on an even keel during the past 365+ days.

Jerry Slaughter has continued to be a most capable Executive Director and a tremendous lobbyist in the Kansas Legislature. Gary Caruthers, as Executive Assistant, withstood the pressures of his duties and remains most dedicated to the Kansas Medical Society and its membership. Val Braun, Executive Assistant, completes the Executive Staff, and maintains her excellent handling of THE JOURNAL's affairs.

The devotion to duty of the members of the Executive Committee has remained quite remarkable during the past year. This has been exemplified by their attending the meetings en masse and freely expounding their views and virtues. Their actions, among others, were:

1. Established a policy that multiphasic health testing should be considered if sponsored and approved by the local county medical society. (This resulted in my receiving letters from nearly every co-op in Kansas inquiring as to my parentage or apparent lack of same.)

2. Established a committee of three physicians to meet with the Kansas Hospital Association on the matter of cost containment.

3. Established a policy that Physician Assistants or extenders should not write prescriptions without some supervision and counter-signature or authorization by a physician.

4. Established a policy on addressographing, stuffing, and mailing services. Members and specialty societies are to be charged $7.50 and non-members $10.00 for these services.

5. Contributed $1,000 to KUMC for Kansas Health Day.

6. Reaffirmed to the AMA that the KMS is opposed to any form of federally controlled health insurance for our nation.

7. Approved an invitation to the president of the Auxiliary of the Kansas Medical Society to serve as an ex-officio member of the Executive Committee.

The Council has taken the following actions during the year.

1. Approved the location of the Annual Meeting for 1979 at Hutchinson, with a slight alteration of the format of the meeting.

2. Endorsed the Workmen's Compensation Program of the Insurance Committee.

3. Reappointed David Gray, M.D., Editor of THE JOURNAL.

4. Accepted the budget as printed.

5. Approved the sum of $8,000 to start and develop proposals for an Impaired Physician Performance Committee.

6. Approved the computerizing of the membership files of the KMS.

7. Reviewed the dues structure and found that due to the frugality of the Executive Staff and the chicanery of your elected officials, it was not necessary to increase your dues for 1978.

The numerous committees (Insurance, Impaired Physician Performance, Cost Containment, Legislation, Medical Education, Welfare, etc.) have all been functioning well. Jackie Burnett, President of the KMS Auxiliary, has been most active during the past year contributing her time, energy, and enthusiasm.

Along with Jackie, I was privileged to help with the installation of Lela May Young as President of the AMA Auxiliary, in San Francisco.

Your delegates and alternate delegates do yeoman work at the AMA House of Delegates meetings. You should see the four of them trying to cover eight or more reference committees, all meeting within a half mile of each other at the same time. The winter meeting of the AMA in Chicago was all business. With a wind chill factor of –60°F outside and nothing but the House of Delegates inside, they had a captive audience.

Jerry Slaughter and I attended the AMA Legislative Meeting in Tarpon Springs, Florida, in January and found that all of the State Societies either were having, have had, or were going to have the familiar problems with brand exchange, chiropractors, physician extenders, legislators, or cost containment.

I was privileged to attend a three-day seminar on negotiations, sponsored by the AMA in Boulder, Colorado, this spring. This is a tremendous learning experience and the AMA is willing to sponsor seminars on negotiations lasting a day and a half if requested, at a nominal fee. I highly recommend that the KMS request a seminar.

I especially would like to thank those individuals who attended the various Council district meetings that we visited. They were indeed the greatest audience that one could hope to find.

I greatly appreciate the privilege of having served as your President this past year. It has been a good year and I thank you for the opportunity.

Executive Director—
Jerry Slaughter

Once again, it is my pleasure to appear before you during the Annual Meeting to give the report of the Executive Director. This is my third appearance as Executive Director, as I am entering my sixth year with the Kansas Medical Society. It seems that the intervening year between meetings goes more quickly than the last, and I am certain with the busy year ahead that this year also will go quickly. One of the true pleasures of being associated with the Medical Society is the opportunity to work with its fine leaders. Doctor Huff has just finished a busy year as President, and one, as you have heard in the earlier reports, that has been a year of accomplishment. Doctor Huff has given unselfishly of his time, and he has been a ready source of inspiration and information during the course of last year. We have had to call on him numerous times for his counsel and advice, and he represented you admirably in the Kansas Legislature. It has been a true pleasure working with this fine man, and I look forward to next year with him remaining on the Executive Committee.

There are many challenges facing medicine today, both nationally and locally. Because this is an election year for state legislators, I hope each and every one of you will take an active interest in the political goings-on, and contribute time, money, or whatever else you may be able to give. I cannot emphasize strongly enough the growing role that each one of you plays in the politics of medicine. Legislators look to their constituents for direction on almost all of the issues that face them during the 90-day legislative session.

As an informed constituent, you are in an ideal position to help influence the changing shape of medical legislation, and I truly hope you will take this opportunity to become involved in the events that will eventually place your representative in the State Capitol.

A significant challenge facing medicine today is the problem of rising health care costs. Although the problem is more often discussed on a national level, the problem, nevertheless, grows more acute right here at home as well. The people of the State of Kansas are looking to the medical profession and the hospital industry for answers, or at least a recognition of this significant problem. This is evidenced by the legislature's unwillingness to pass two bills which would have established mandatory rate review commissions, opting instead for a route that would give the private industry a chance to demonstrte some cost reduction. The Kansas Medical Society and the Kansas Hospital Association have joined a national effort, called the Voluntary Effort, to help identify and reduce the increasing health costs, especially in hospitals. I am not certain there are any easy solutions to these problems, but medicine must at least make a legitimate, sincere attempt to get a handle on the problem of rising health costs.

In short, I believe the coming year will be one of challenges and new issues. The Society will be in good

hands as Dr. Warren Meyer assumes the post of President at the end of this meeting. Doctor Meyer is a man of integrity and capability, and I know he is well qualified to face the important tasks ahead.

We have had a couple of new additions to the staff since last year. Eleanor Bell is assisting Val in the production of THE JOURNAL, and doing an admirable job. Ramona Perez has just been added to the staff to fill Velma White's position, and Ramona is doing a fine job implementing our new computer-assisted membership files. We have also added just recently a new secretary, Lee Barber, who is a typist par excellence, and a welcome addition to the Medical Society office.

The two people who make my job easy from time to time, and really help keep the work flowing smoothly are Val Braun and Gary Caruthers. The energy, expertise and abilities of these two people are a real asset to me, and I consult with them frequently on society affairs. I feel most fortunate to have Val and Gary, and I believe that they are the finest anywhere in the country. I simply could not do the job without their able assistance.

I would like to take this opportunity to express my sincerest thanks to all of you for the confidence you have shown in me during the last few years. I find working with the Kansas Medical Society to be extremely rewarding and a most pleasurable experience. It is a privilege to be associated with this organization, and I look forward to continuing that relationship. Thank you.

SECOND SESSION

The second session of the House of Delegates was called to order by the Speaker, Clair C. Conard, M.D., at 9:00 AM on Wednesday, May 10, 1978, at the Downtown Ramada Inn, Topeka.

The Speaker placed some rules before the body, and announced that Sturgis Standard Code of Parliamentary Procedure would be followed at this meeting. Every delegate will be given an opportunity to be heard on every question, but except for the person who makes the motion, all delegates were asked to cooperate by being heard only once upon a single question.

Ballots for the election of officers were distributed, and the following results reported:

PRESIDENT-ELECT: Donald D. Goering, M.D., Salina

FIRST VICE PRESIDENT: Phillip A. Godwin, M.D., Lawrence

SECOND VICE PRESIDENT: Herman W. Hiesterman, M.D., Quinter

CONSTITUTIONAL SECRETARY: Jack R. Cooper, M.D., Shawnee Mission

TREASURER: William K. Walker, M.D., Sedan

AMA DELEGATE 1979/80: Alex Scott, M.D., Junction City

AMA ALTERNATE DELEGATE 1979/80: Kermit G. Wedel, M.D., Minneapolis

SPEAKER: Clair C. Conard, M.D., Dodge City

VICE SPEAKER: John O. Yulich, M.D., Kansas City, KS

The following results of Council district elections were announced:

DISTRICT 1—Roger D. Warren, M.D., Hanover

DISTRICT 3—LEWIS G. ALLEN, Jr., M.D., Shawnee Mission

DISTRICT 5—Kenneth M. Boese, M.D., Manhattan

DISTRICT 8—Steven J. Smith, M.D., Arkansas City

DISTRICT 9—Herbert D. Doubek, M.D., Belleville

DISTRICT 17—Max E. Teare, M.D., Garden City

William R. Lentz, M.D., Topeka, Chairman, was asked to report on the activities of the Insurance Committee.

Dr. Lentz described in detail the Kansas Medical Society Life Insurance Program. During the enrollment period (now until August 1) any member of the Society, whether insurable or not, may purchase an individual policy which remains in force even if the person were to terminate his medical practice or leave Kansas. Those uninsurable are limited to coverage up to $50,000. Policies of up to $500,000 are available, with evidence of insurability for amounts over $100,000 required.

Physicians' offices will be contacted by the insurance agency, Dr. Lentz said. At least 360 persons must be enrolled in Kansas for this program to be offered here. The rates are competitive group rates, and Dr. Lentz strongly recommended this program.

William C. Swisher, M.D., Wichita, Member of the Kansas State Board of Healing Arts, addressed the House concerning the CME requirements for relicensure effective July 1, 1978.

A recent mailing to the physicians from the Healing Arts Board contained the following items:

1. A green questionnaire developed by the Department of Health regarding data of medical practice in Kansas;

2. An affidavit for exemption from the necessity to fulfill the CME requirements by those who are retired and are not engaged in active practice of medicine;

3. A list of certifying agencies accepted in Kansas for CME accreditation.

Judging by the number and tone of inquiries received by the Board, Dr. Swisher said that apparently many

physicians are not informed on the subject of CME requirements. He asked the delegates present to impress on physicians in their districts the importance of becoming educated on this topic. The Healing Arts Board is not empowered to grant exemptions; it is limited by law and must withhold the license of any person who does not meet the Continuing Medical Education requirement.

Dr. Huff handed the gavel to the incoming President, Warren E. Meyer, M.D., Wichita. Dr. Meyer delivered a brief address and announced the following chairmanships:

David E. Gray, Topeka—Editorial Board

Daniel K. Roberts, Wichita—Professional Liability/ Medical-Legal Affairs

Antonio Huaman, Topeka—KaMPAC

Jimmie A. Gleason, Topeka—Legislative Committee

Emerson D. Yoder, Denton—Mediserve

Lew W. Purinton, Wichita—Education

George M. Penn, Topeka—Impaired Physician Program

Kermit G. Wedel, Minneapolis—Health Services

Ivan E. Rhodes, Wichita—Sociology and Economics

Joseph E. McMullen, Hutchinson—State Meeting

Other appointments will be made shortly, in accordance with the newly passed Resolution No. 78-1.

Dr. Meyer announced that the Society is in sound condition and that the Executive Committee and Council will continue to do all in their power to maintain this state. He listed the following as some of the most important areas of involvement for the coming year:

Legislation. The Legislative Committee and the Key Man program will continue to be very active. This being an election year, Dr. Meyer called for greater involvement in KaMPAC. Funds are vital especially on the state level.—The Interim Legislative Committees will continue to meet during the summer. KMS must and will be involved in the deliberations on the credentialing of health care.—Reorganization of the Healing Arts Board debate is coming up. KMS will testify in this area.—Annual problems of Medicaid, Medicare, naturopaths, chiropractors and others will continue to surface.—Organized medicine must continue to be involved in HSAs. While there is little opportunity for direct involvement because of the wording in the law, some parallel committees can be established to monitor the work of these agencies.—On the national scene, NHI is a possibility. It may be necessary to mobilize resources as was done in the professional liability effort, to promote public education in this area.

Impaired Physician Program. The Kansas Medical Society has developed a program to rehabilitate physicians. Rather than punish them—unless all avenues have failed or public health is endangered—the Society is interested in restoring the qualified practitioners to the profession. More effort will be expended in this field.

State Board of Healing Arts. Cooperation with this body is essential. There are still problems with CME, but they are being resolved. The Education Committee will meet with those who experience difficulties. There are increasingly more facilities offering accredited education in Kansas, so that obtaining the necessary number of hours should not pose an undue hardship.

Physician Involvement. Perhaps the area of greatest deficiency in Society work is physician involvement in the affairs of organized medicine. Dr. Meyer called on all those present to encourage physicians in their districts to become involved in the affairs of the Society: "If there are those who do not approve of our efforts, it is their responsibility to tell us about it." The Society is concerned about all of the physicians and needs their input. More goals can be reached for the good of Kansans by a unified organization. All efforts at neutralizing and fragmenting the profession and its influence must be resisted.

Dr. Meyer announced that he intends to visit as many component medical societies as will invite him to come. He hopes all societies in Kansas will do so. He then closed his address with a prayer.

The Speaker reminded the House that the 1979 Annual Session will be held at the Holiday Inn Holidome, Hutchinson. The format has been changed, and the meeting will be shorter. He announced the following schedule for 1979:

Thursday, May 3—Sports Day
Friday, May 4—First House of Delegates
Saturday, May 5—Scientific Program
Sunday, May 6—Second House of Delegates

Dr. Conard complimented the delegates on the skillful and calm manner of deliberations, and asked them to encourage their colleagues to become involved. He announced that the Council will meet immediately following the adjournment of this House.

The meeting adjourned at 11:45 AM.

An asterisk following the resolution number indicates that those resolutions require a change in the Constitution and By-Laws.

RESOLUTION NO. 78-1*

Commissions and Committees

Resolved, That Sections 9.10 through 9.212 in-

clusive be stricken from the By-laws; and be it further

Resolved, That the following be inserted therein: 9.0—Commissions and committees are appointed by the President for a one-year term. The Chairman, size, and membership of such commissions and committees shall be designated by the President. Commissions and committees may introduce resolutions for consideration by the House of Delegates; and be it further

Resolved, That the By-laws be renumbered to reflect the above changes.

RESOLUTION NO. 78-2*

Out-of-State Members—CME

WHEREAS, The Kansas Medical Society is primarily interested in medicine in Kansas; and

WHEREAS, The continuing medical education requirement for membership now applies to all active members; and

WHEREAS, Kansas State Board of Healing Arts will exempt out-of-state physicians from the CME relicensure requirement; therefore be it

Resolved, That the By-laws be amended to exempt out-of-state members from the continuing medical education requirement for membership; and be it further

Resolved, That this resolution be forwarded to the Committee on Constitution and By-laws to draft the appropriate language.

RESOLUTION NO. 78-3

Verification of CME

WHEREAS, The Kansas State Board of Healing Arts has adopted a mechanism other than the AMA Physician's Recognition Award and specialty society CME certification, for verification of the continuing medical education requirement for relicensure; therefore be it

Resolved, That the Kansas Medical Society, in addition to the AMA Physician's Recognition Award and specialty society CME certification, accept the mechanism established by the Kansas State Board of Healing Arts as proof of CME for continued membership.

RESOLUTION NO. 78-4*

Retirement Membership Status

WHEREAS, The continuing medical education requirement for relicensure is an extraordinary burden upon the retired physician; and

WHEREAS, Some retired physicians are relinquishing their license to practice medicine rather than to comply with the requirement; and

WHEREAS, A physician may not be a member of the Kansas Medical Society without a license to practice medicine; therefore be it

Resolved, That Sec. 1.6122 of the By-laws be amended to make provision for retirement membership status in the Kansas Medical Society without a license to practice medicine; and be it further

Resolved, That this resolution be forwarded to the Committee on Constitution and By-laws to draft the appropriate language.

RESOLUTION NO. 78-5

Professional Liability Insurance—Retired Physicians

WHEREAS, Kansas law requires physicians to carry professional liability insurance as a requisite for licensure; and

WHEREAS, Physicians not in the active practice of the healing arts may sign an affidavit exempting them from the continuing medical education requirement for relicensure; therefore be it

Resolved, That the General Counsel of the Kansas Medical Society review the possibility of a similar exemption from the professional liability insurance requirement for relicensure; and be it further

Resolved, That the Executive Committee take appropriate action based on the findings of the General Counsel.

RESOLUTION NO. 78-6*

Kansas Chapter, American College of Emergency Room Physicians

WHEREAS, The Kansas Chapter, American College of Emergency Room Physicians is duly organized and represented by 61 physicians in Kansas; and

WHEREAS, The Council of the Kansas Medical Society has reviewed the application for specialty

society status and recommends approval by the House of Delegates; therefore be it

Resolved, That Sec. 4.58 of the By-laws be amended with the addition of 4.58,19 "Kansas Chapter, American College of Emergency Room Physicians."

RESOLUTION NO. 78-7

Joint Billing

WHEREAS, The Kansas Medical Political Action Committee (KaMPAC) has achieved a notable record of success in electing friends of Kansas medicine to public office at the state and federal level; and

WHEREAS, KaMPAC has proven to politicians and governmental bureaucrats alike that the Kansas Physician Community is a powerful force which must be reckoned with; and

WHEREAS, Government encroachment in the health care system must be dealt with through increasingly effective means of political action; therefore be it

Resolved, That the county and component medical societies be encouraged to include a provision for KaMPAC dues on their membership billing statements.

RESOLUTION NO. 78-8

Direct Dues Billing

Resolved, That the Executive Office, upon request and when feasible, assist component societies in the billing for and collection of annual dues.

RESOLUTION NO. 78-9*

Lincoln, Rush, and Ness Counties

WHEREAS, Lincoln, Rush, and Ness counties are now included in the Central Kansas Medical Society; therefore be it

Resolved, That Sec. 8.49 of the By-laws be changed to delete Lincoln County; and be it further

Resolved, That Sec. 8.4,14 of the By-laws be changed to delete Rush and Ness Counties; and be it further

Resolved, That Sec. 8.4,13 of the By-laws be changed to include Lincoln, Rush, and Ness counties.

RESOLUTION NO. 78-10*

Bylaws Change, Chautauqua-Elk Counties

WHEREAS, Resolution 76-7 established Council District 19 consisting of the following counties: Allen, Chautauqua, Elk, Montgomery, Neosho, Wilson and Woodson: therefore be it

Resolved, That Sec. 8.48 of the Bylaws be amended by deleting Chautauqua and Elk counties.

RESOLUTION NO. 78-11

HR-1818

Resolved, That the Kansas Medical Society urge the American Medical Association to withdraw support of HR-1818.

RESOLUTION NO. 78-12

Not adopted. Tabled.

Federal Regionalization

WHEREAS, The United States recently has been divided into ten (10) federal regions by Executive Order 11647-Federal Regional Councils, entered in the *Federal Register* by the President on February 12, 1972; and

WHEREAS, The Federal Regional System will assume the power and authority which traditionally has belonged to municipal and county governments and even to sovereign states, and will bypass the traditional and Constitutional governmental bodies; and

WHEREAS, Across the nation, this new form of government is being created under different names such as Mid-America Regional Council, River Basin Compacts, Metropolitan Regions, County Consolidated Charters, and Federal Region—Sub-state regions, all of which constitute "metro" or "regional" government; and the concept of regionalism is also being advanced through the Health Systems Agencies being created under the Health Planning Act; and

WHEREAS, The direct application of these principles through "MASHA" to the general and private practice of medicine is of doubtful value and is oppressive; and

WHEREAS, A consequence of federal regionalism will be further eroding the rights and freedoms of all Kansans guaranteed by the Constitution of the

State of Kansas, and of the United States; and

WHEREAS, Senators and Representatives of the Kansas Legislature have taken an oath to uphold the Constitution of Kansas and of the United States, and, therefore, must hold as sacred trust their responsibility to preserve and protect the freedom and liberties of citizens of Kansas; and

WHEREAS, The Johnson County Medical Society has submitted a similar resolution in 1977 and again desires to bring this resolution before the House of Delegates of the Kansas Medical Society; therefore be it

Resolved, That the House of Delegates of the Kansas Medical Society in regular session assembled in Topeka, Kansas, this 10th Day of May, 1978, urges the Governor of Kansas and the Kansas Legislature to establish a special joint commission consisting of four senators and four representatives as members of a standing commission and that they consider the following points:

1. That, in order that an open and objective investigation may be performed, the chairman shall *not* be someone who has already committed himself to the advancement of regional government;

2. That said commission shall thoroughly investigate regional government in all its aspects and under whatever name or form, including, but not limited to, its origins, developments, functions, purposes and goals, and shall take testimony, hold public hearings, invite expert and interested citizen witnesses, and take whatever action is necessary to carry out this joint resolution (Senate and House);

3. That said commission shall report its findings and recommendations, especially as to the constitutionality of regional government and its effects upon the citizens of Kansas by July 4, 1979; and, in addition, shall report its findings and recommendations together with any proposed legislation deemed necessary to protect and preserve the independent statehood of Kansas and the rights of Kansas citizens;

4. That the Kansas Legislature make all of their authority, resources, and influence available without reservation to preserve, protect, and defend Article X, the Bill of Rights of the Constitution of the United States which declares:

"The powers not delegated to the United States by the Constitution nor prohibited by it to the states, are *reserved to the states* respectively, or *to the people.*" (Emphasis added)

RESOLUTION NO. 78-13

Medical Practice

WHEREAS, The indication for the use of a medicine is a medical not a political judgment; and

WHEREAS, Medical indications change depending upon research and medical progress; and

WHEREAS, There is a growing tendency for governmental units to attempt to restrict the use of medicines on other than medical basis; therefore be it

Resolved, That all component medical societies and individual physicians be encouraged to educate their legislators as to the dangers of attempting to promote or restrict, by law, the use of legitimate medicines; and be it further

Resolved, That this resolution be introduced at the AMA Annual Meeting in St. Louis, Missouri, in June 1978, and that copies of this resolution be sent to Kansas representatives in Congress and to members of Kansas Legislature.

RESOLUTION NO. 78-14

Locum Tenens

WHEREAS, The Kansas Medical Society has established a locum tenens clearinghouse; and

WHEREAS, It has been determined that current residency programs do not allow residents adequate time to participate in locum tenens; therefore be it

Resolved, That the Kansas Medical Society request the Residency Program Review Committees to consider allowing at a minimum one week per year per resident for locum tenens, and that this time be considered as educational value towards completion of the residency program; and be it further

Resolved, That a copy of this resolution be forwarded to the American Medical Association for consideration on the national level.

RESOLUTION NO. 78-15

Prescriptions by Allied Health Personnel

WHEREAS, As physician assistants, nurses, and other allied personnel assume a larger role in the delivery of health care, there is increased pressure to allow such allied personnel to write prescriptions; and

WHEREAS, Only physicians and dentists possess

the necessary background and training which is required to be able to prescribe in the safest manner; therefore be it

Resolved, That the Kansas Medical Society oppose permitting the allied health personnel to initiate and write prescriptions.

RESOLUTION NO. 78-16

Committee for the Impaired Physician Program

WHEREAS, The Kansas Medical Society believes that the professional problems of the impaired physician are the responsibility of the medical profession; and

WHEREAS, The Kansas Medical Society is motivated in this area by humanitarian concerns for the public and the impaired physician and the family; and

WHEREAS, The Kansas Medical Society recognizes that alcoholism, other drug abuse, mental, physical and aging problems, and/or medical incompetence may represent forms of physician impairment; and

WHEREAS, The above conditions may be alleviated if recognized and treated; and

WHEREAS, The Kansas Medical Society Resolution No. 77-7 directed the Society to undertake a study of possible solutions for the problem of physician discipline in the State of Kansas; and

WHEREAS, The Kansas Medical Society's legislative effort in 1978 resulted in the passage of HB-3109, authorizing the Kansas State Board of Healing Arts to invoke temporary suspensions of licenses in appropriate cases; therefore be it

Resolved, That the Committee for the Impaired Physician Program be directed to implement the proposed statewide Impaired Physician Program; and be it further

Resolved, That the Committee for the Impaired Physician Program work in liaison with the State Board of Healing Arts as the formal agency with the power to deal with disabled physicians; and be it further

Resolved, That all actions taken in the area of impaired physicians in the Kansas Medical Society are intended in the best interests of the physician and the public, and will be done in such a manner as to preserve unimpaired the physician's right to continue practice without restriction or stigma on recovery or remission.

KMS IMPAIRED PHYSICIAN PROGRAM

Statement of Purpose

We believe that the problem of impaired professional performance among physicians is the responsibility of the medical profession, requiring us to deal with such physicians unequivocally, fully, and compassionately with all appropriate means at our disposal. To meet this need, we recommend that a committee be established along with a comprehensive action program.

The purpose of the Committee for the Impaired Physician Program is to:

1. Establish a statewide non-coercive program for locating, contacting, and offering rehabilitative help to physicians who have become professionally impaired to varying degrees because of alcoholism, other drug dependence, mental, physical and aging problems, and/or medical incompetence.

2. Function as a peer review organization.

3. Continue to work in liaison with the State Board of Healing Arts as the formal agency with the legal authority to deal with the impaired physician.

4. Establish programs of education and prevention concerned with alcoholism, other drug dependence, mental, physical and aging problems, and/or medical incompetence.

General Principles

Regarding the impaired physician, the Kansas Medical Society subscribes to the following guiding principles:

1. We are motivated in this area by humanitarian concern for the public and the impaired physician and his family.

2. We recognize that alcoholism, other drug dependence, mental, physical and aging problems, and/or medical incompetence represent forms of physician impairment that may be ignored or untreated.

3. We recognize that most alcoholism, other drug dependence, mental, physical and aging problems and/or medical incompetence are treatable conditions.

4. As practitioners of the healing arts, we, more than any other group, should favor treatment and/or rehabilitation of our impaired colleagues above any other alternative.

5. We encourage all impaired physicians to seek help and cooperate in treatment by all means at the disposal of the Kansas Medical Society.

6. We favor the *earliest possible* intervention in cases of physician impairment while personal, financial, mental, and physical resources are intact and minimum damage has been done to professional reputation or to the public.

7. All Kansas Medical Society action taken on behalf of the impaired physician will be done in such a manner as to preserve, unimpaired, the physician's right to continue practice without restriction or stigma upon recovery or remission.

8. All actions taken in the area of impaired physicians by the Kansas Medical Society are intended in the best interest of the physician and the public.

9. Referral to the State Board of Healing Arts for sanctions will be considered only when the impaired physician's actions endanger the public or himself and he repeatedly refuses assistance. When a referral is made to the Kansas State Board of Healing Arts by the Committee for the Impaired Physician Program, the State Board of Healing Arts will act to suspend the license of the physician in question according to the laws governing such licensure. Any temporary suspension or limitation order would take effect when served in person on the licensee.

Structure

I. The Impaired Physician Program of the Kansas Medical Society will encompass the entire State of Kansas and be administered by the Kansas Medical Society. Existing mechanisms on a local level will be integrated and utilized by the statewide program, but the main impetus for continuing the program must come from the state society.

II. There will be established two committees: the Committee for the Impaired Physician Program and the Physician Contact/Support Committee.

A. *The Committee for the Impaired Physician Program:* This Committee will consist of ten (10) physicians who are knowledgeable and vitally interested in the subject of physician impairment. Additional members may be added at the discretion of the Committee for a designated length of time. Appointed terms of adequate length should be established to ensure continuity; additionally, the appointments should be on a rotating basis. The Committee should be established as a standing committee of the Executive Committee of The Kansas Medical Society and accountable to the executive committee.

The Committee for the Impaired Physician Program will be responsible to:

1. Broadly publicize the Impaired Physician Program and direct an educational effort to physicians and their families, hospital staffs, county medical societies, auxiliary, and others to communicate the general principles of the program and solicit cooperation;

2. Establish a registry of the best appropriate resources and therapists in the areas of alcoholism, other drug dependence, mental health, geriatrics, and/or medical incompetence. This registry will be continually updated as necessary and will serve as the basis for designating intervention teams;

3. Supply current factual information concerning these conditions and be able to make knowledgeable referrals for effective treatment.

4. Confirm complaints of physician impairment and then refer to a Contact/Support team for motivation into voluntary treatment;

5. Evaluate the quality and efficiency of services rendered by physicians referred to the Committee for the Impaired Physician Program;

6. Recommend overall policy and develop guidelines regarding the implementation and maintenance of the Impaired Physician Program;

7. Establish criteria for participating as a Contact/Support team member and be responsible for quality control of this membership;

8. Require follow-up information to be provided by the Contact/Support teams to determine the current status of the impaired physician who has begun treatment;

9. Conduct routine administrative functions including fiscal management and recommendation of needed budget;

10. Make recommendations to the State Board of Healing Arts regarding any impaired physician when a majority of the Committee agrees that the reasonable efforts of the Contact/Support teams have failed to produce an acceptable degree of rehabilitation.

B. *Physician Contact/Support Committee:* There will be established a larger group from which Contact/Support team members are drawn. The names of these people should be maintained by the Committee for the Impaired Physician Program in a resource registry. Contact/Support team members should be drawn from all areas of the state, should have expertise in the areas of alcoholism, other drug dependence, mental health, geriatrics and/or medical incompetence and some should themselves be rehabilitated impaired physicians. When the Committee for the Impaired Physician Program deems it appropriate, a Contact/Support team will be selected in the following manner: Where evidence exists that a physician's ability to function professionally is impaired, the Committee will select a Contact/Support team. At least one member of this team will be a physician from the resource registry. The registry may be the source of the entire team. No member of the team will be involved in the ongoing treatment process.

It is often advisable that physician Contact/Support members be drawn from a locale outside of the geo-

graphic area of the impaired physician's practice.

It will be the responsibility of the Contact/Support team to:

1. Meet with family members and others in appropriate settings, with the impaired physician and present him with documentation that impairment exists; and to express the concerns of his colleagues and family;

2. Encourage the impaired physician to seek help voluntarily, or assist him into active treatment;

3. Maintain contact with the impaired physician to support and encourage his cooperation in the treatment and in the case of the alcohol and/or other drug dependent physician for active participation in Alcoholics Anonymous; and encourage membership in International Doctors in Alcoholics Anonymous (IDAA), and the American Medical Society on Alcoholism;

4. Make every effort to assist the impaired physician to continue his professional duties insofar as he is considered able to do so by the physician in charge of treatment;

5. Attempt to plan for, or to help, provide for opportunities for the impaired physician to continue limited duties, supervised if necessary, as thought reasonable by his treating physician;

6. In cases where the impaired physician has discontinued or limited his work, aid him to return to appropriate work duties as soon as possible;

7. Where there is a failure to seek treatment and/or failure to cooperate after a reasonable period of time, this fact will be reported to the Chairman of the Committee for the Impaired Physician Program.

III. Referral calls concerning any alleged impaired physician shall be made to the Kansas Medical Society Headquarters in Topeka. Call collect (913) 235-2383. This number will be answered 24 hours a day.

A person reporting an alleged impaired physician will be asked: (1) the name of the alleged impaired physician; and (2) the general nature of the complaint. The caller will be asked to identify himself if he wishes, but will be assured that his complaint is confidential and that his name will not be revealed to the alleged impaired physician without the caller's knowledge and consent.

Following receipt of any call, the Kansas Medical Society office staff will contact the Chairman of the Committee for the Impaired Physician Program, or his designee, and report the basic information obtained from the complainant. The Chairman or his designee will telephone an appropriate member of the county medical society in the alleged impaired physician's Council district to begin the process of verification and/or assessment of the need to pursue the complaint with a Contact/Support team.

A. *Voluntary or Self-referral:* When the impaired physician contacts the Kansas Medical Society program directly, the following steps will take place:

1. The appropriate staff member receiving the call contacts the Chairman or his designee of the Committee for the Impaired Physician Program;

2. The Chairman or his designee arranges for a member of this Committee to contact the impaired physician to inquire about the nature of his illness or problem, and to discuss with the physician his willingness to undertake appropriate treatment;

3. The Chairman or his designee then assigns a Contact/Support team who will become advocate for the physician as he undergoes treatment. The steps outlined under the "responsibilities of the Contact/Support team," (points 3-7), will then go into effect.

B. *Involuntary or Other Means of Referral:* The Kansas Medical Society will accept from any responsible source (physician peer, hospital administrator, patient, physician's spouse, etc.) information indicating that a physician may be impaired. *Specific details regarding the physician involved will not be sought or accepted at the time of the original call.* In such a case, the following steps will take place:

1. The appropriate staff member receiving the call contacts the Chairman of the Committee for the Impaired Physician Program, or his designee; and reports: (1) the name of the alleged impaired physician; (2) the general nature of the complaint; and (3) the caller's name and telephone number if volunteered by the caller.

2. The chairman or his designee contacts an appropriate member(s) of the county medical society in the alleged impaired physician's council district to inquire about the alleged impaired physician along with the specific reasons for the alleged impairment.

3. The Committee member then evaluates, with the assistance of other members of the Committee as he deems necessary, the information, making as much use as possible of firsthand information concerning the specific incidents of the demonstrated impairment. All such information gathered by the Committee for the Impaired Physician Program will be kept confidential.

4. Having determined sufficient cause exists to justify contacting the physician thought to be impaired, the Committee by direction of its Chairman or his designee will select a Contact/Support team. Steps 1-7 under "responsibilities of the Contact/Support team," then go into effect.

C. *Conclusion of Intervention.* When a physician re-

ferred to the Kansas Medical Society is considered rehabilitated, and restored to medical practice, the Kansas Medical Society intervention is concluded. This may require a period of anywhere from 2 to 3 or more years. All records are then destroyed.

When all efforts fail and the degree of impairment of the physician's professional performance threatens the public trust, the matter will be reported to the Kansas State Board of Healing Arts by the Chairman of the Committee for the Impaired Physician Program.

Legal Issues

1. *Immunity from Liability:* Exemption from liability for all those who are actively involved in the program, including those who in good faith report cases of physician impairment, the Kansas Medical Society committees for the Impaired Physician Program and the Contact/Support Teams, the Kansas Medical Society itself, both generally and including the Executive Committee of The Kansas Medical Society, as well as any of the committees, staff, counsels, or commissions of this Society otherwise involved in the program, is provided through the following mechanisms:

KSA 65-2898 and KSA 65-4909 provide immunity by relieving any person reporting to the State Board of Healing Arts and other licensing boards from civil liability for reporting any alleged incidents of malpractice or the qualifications, fitness or character of persons licensed or registered by such boards, if such report is made under oath and in good faith.

The bills also provide that any state, regional, or local association composed of persons licensed to practice the healing arts, or other health care providers, or any individual members or committees thereof which investigate and communicate information pertaining to alleged malpractice incidents or the qualifications, fitness or character of any licensee or registrant to the appropriate licensing board, shall be immune from liability in civil actions if the investigation and communication were made in good faith and did not represent as true any matter not reasonably believed to be true. On this basis, members of the Committee for the Impaired Physician Program and the physician Contact/Support Committee, as well as others reporting, are granted immunity, so long as the conditions of the statute are met.

2. *Confidentiality:* The purpose of the Kansas Medical Society program for impaired physicians is to offer early intervention and help for impaired physicians, at the same time performing a peer review function by evaluating the quality and efficiency of services rendered by physicians referred to the Committee for the Impaired Physician Program. The entire program should be carried out with minimal disruption of the physician's practice, and no harm to his reputation. For these reasons, all information gathered on the physician in question must be held in the strictest confidence and any investigation of reports carried out discreetly. Proper records must be maintained but only in strictest confidence and protection.

3. *Relationship to the State Board of Healing Arts:* The Kansas Legislature enacted HB-2008 in 1975, which grants the Healing Arts Board the authority to utilize the AMA Model Act on the impaired physician. This act was drafted by AMA in 1974, and allows each individual state board considerable latitude and authority to act in the area of physician impairment. It is clear that the trend seems to be in the direction of closer cooperation between state medical associations and licensing bodies along the entire continuum of detection, diagnosis, treatment and rehabilitation.

HB-3109, enacted in 1978, grants the Healing Arts Board authority for temporary suspension and limitation of license of any licensee, without notice or hearing, if the Board determines that there is cause to believe that grounds exist for such revocation, suspension or limitation, and that the licensee's continuation in practice would constitute an imminent danger to the public health and safety. Any temporary suspension or limitation order would take effect when served in person on the licensee.

The Board will institute proceedings for a hearing simultaneously with the Board's action to limit or suspend the license temporarily. Such hearing is to be held within fifteen (15) days from the date of the temporary limitation or suspension unless a continuance is requested by the licensee.

In no case shall a temporary suspension or temporary limitation of a license be in effect for a period of time in excess of 90 days. At the end of such period of time, the licensee shall be reinstated to full licensure unless the Board has revoked, suspended or limited the license.

With the support and cooperation of the State Board of Healing Arts, the Kansas Medical Society's program includes non-member physicians. The alternative for the impaired non-member would be either access to the Kansas Medical Society's mechanism or in the event of failure to accept this help, referral to the State Board's formal mechanism. With the State Board of Healing Arts in the background as the "court of last resort," the Kansas Medical Society's efforts should be more effective in the great majority of instances of physician impairment.

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Mary Ann Penn, R.N., Auxiliary Liaison	Topeka

RESOLUTION NO. 78-17

Voluntary Effort-Cost Containment

WHEREAS, The American Hospital Association, the American Medical Association, and the Federation of American Hospitals, in recognition of the need to restrain the rate of increase in health care expenditures, agreed to organize a Voluntary Cost Containment Program and urge each hospital to reassess its operating and capital budgets to see if anything further can be done to reduce these budgets, consistent with sound medical practice; and

WHEREAS, The American Hospital Association, the American Medical Association, and the Federation of American Hospitals have organized a National Steering Committee on Voluntary Cost Containment, which has developed a 15-point program to be implemented through state-level voluntary cost containment committees and through voluntary action of individual hospitals; and

WHEREAS, The Kansas Cost Containment Committee has been organized to develop and carry out a voluntary action program that will contribute, to the maximum extent possible, to the goals and objectives of the National Steering Committee; and

WHEREAS, The Kansas Voluntary Cost Containment Committee has reviewed the program of the National Steering Committee and has endorsed in principle its goals and objectives; therefore be it

Resolved, That the Kansas Medical Society supports the nationwide activities of the Voluntary Effort to reduce the rate of increase in health care costs, consistent with sound medical practice, and will develop and carry out ongoing voluntary action in the state that will contribute, to the maximum extent possible, to the goals and objectives of this program.

RESOLUTION NO. 78-18*

Resident Physicians' Section

Resolved, That a Section on Resident Physicians be established in the Kansas Medical Society, and the By-Laws be amended as follows:

1. A new section 4.58,20, *Resident Physicians Society,* be added to the By-Laws; and
2. That the By-Laws be further amended in the following manner: Section 5.22—Organized Specialties—Each organized specialty section recognized by the Society shall be entitled to one (1) delegate and one (1) alternate to be chosen by the section.

RESOLUTION NO. 78-19

Cost of Health Care—Impact Statement

Not adopted.

RESOLUTION NO. 78-20

Cost of Health Care—Public Agencies

Resolved, That the Kansas Medical Society believes that all capital expenditures of public money in the health care field should be subject to the provisions of Public Law 93-641, including Certificate of Need requirements; and be it further

Resolved, That the cost accounting procedures for determining health care expenditures should include those expenditures made by government, as well as by private industry; and be it further

Resolved, That copies of this resolution be forwarded to members of Kansas Legislature, Kansas representatives in Congress, Governor Robert Bennett, Secretary Robert Harder, Secretary Dwight Metzler, and the State Health Coordinating Council.

RESOLUTION NO. 78-21*

Nominating Committee

Resolved, That Section 6.6 of the Bylaws be changed as follows: In the sentence, "two (2) or more candidates for the office of delegate-elect, and alternate delegate-elect to the American Medical Association," delete "two (2)" and insert "one (1)."

RESOLUTION NO. 78-22

Not adopted. Referred to the Executive Committee.

Advertising

WHEREAS, The Federal Trade Commission, within the past year, has issued utterances relative to the question of advertising by members of the licensed professions; and

WHEREAS, These utterances have created confusion and raised questions concerning the interpretation of and relationship to the medical profession's Code of Ethics and the Kansas State Board of Healing Arts Act; and

WHEREAS, There is a need to clarify the concept of advertising as differentiated from the concept of solicitation of patients as it relates to the medical profession; therefore be it

Resolved, That the Kansas Medical Society request that the Kansas State Board of Healing Arts, in consultation with the appropriate representatives of the Kansas Medical Society, clarify and develop guidelines concerning advertising as it relates to the practice of medicine in the State of Kansas.

RESOLUTION NO. 78-23

Physician Reimbursement by Third Parties

WHEREAS, The majority of insurance companies providing medical care coverage do not have reimbursement agreements with physicians and thus base their payments on undefined, unilaterally derived formulas; and

WHEREAS, In many instances, the insurance company's fee allowance is less than the physician's professional charge; and

WHEREAS, In those instances where the physician's fee is more than the determined allowance, the insurance company, in its explanatory correspondence to the patient, often subtly implies that the physician's charge is unreasonable and out of line with the charges of physicians of like training practicing in the locality; and

WHEREAS, At a time when medical care costs are of a concern to all, this type of correspondence, using cleverly worded phrases, casts shadows of impropriety on the profession; therefore be it

Resolved, That the Kansas Medical Society voice its opposition to the use of such explanatory letters which are unfair and untruthful by innuendo; and be it further

Resolved, That this entire question be referred to the appropriate committee of the Kansas Medical Society for review and discussion with the Insurance Commissioner of the State of Kansas.

RESOLUTION NO. 78-24

Shawnee County Medical Society

WHEREAS, The Shawnee County Medical Society has hosted an excellent meeting and brought together a group of speakers and programs that were timely and informational; be it

Resolved, That the Kansas Medical Society extend its thanks to the Shawnee County Medical Society and its Auxiliary for their efforts in our behalf.

Council Meeting

(Continued from page 267)

The Council approved a medical component of the Kansas Cavalry, to be used for recruitment of physicians for Kansas.

All previous actions of the Executive Committee were approved.

Doctor Meyer announced that he intends to schedule Council meetings at different locations over the state. The Executive Committee meetings will be held on third Thursdays, unless a more convenient time can be found.

The Council adjourned with a luncheon.

MOVING?

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CME Relicensure in Kansas

Reporting Date Is Near—July 1, 1978

From and after July 1, 1978, every licensee rendering health care service in the state is required to submit with his or her request for license renewal evidence of satisfactory completion of a program of continuing education.

The Board of Healing Arts will accept completion of the AMA Physicians Recognition Award or the CME certification programs of those specialty societies recognized as equivalent to the Physicians Recognition Award.

Upon completion of the CME requirement, the AMA or the specialty society will notify the Kansas Medical Society office. A certificate will be mailed to you prior to your licensure renewal date. Please retain one copy of the certificate for your files and forward one copy to the Board of Healing Arts with your license renewal form.

Any physician licensed after July 1, 1975, will have three years to complete 150 hours of continuing medical education for licensure renewal.

If an applicant for renewal will not render health care service in Kansas, the applicant shall submit with his or her request for license renewal an affidavit stating that he or she will not render such health care service during the term of the license.

The Board of Healing Arts may grant a waiver for a specified period not to exceed eight (8) months to a physician who has suffered an illness or accident making it impossible or extremely difficult to comply with the CME requirement.

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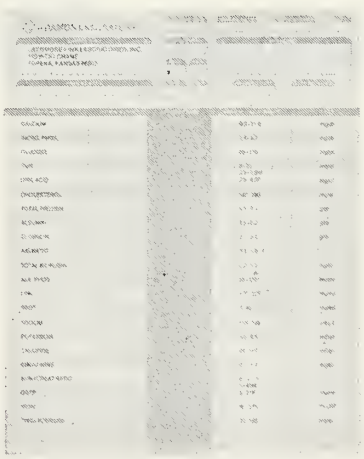
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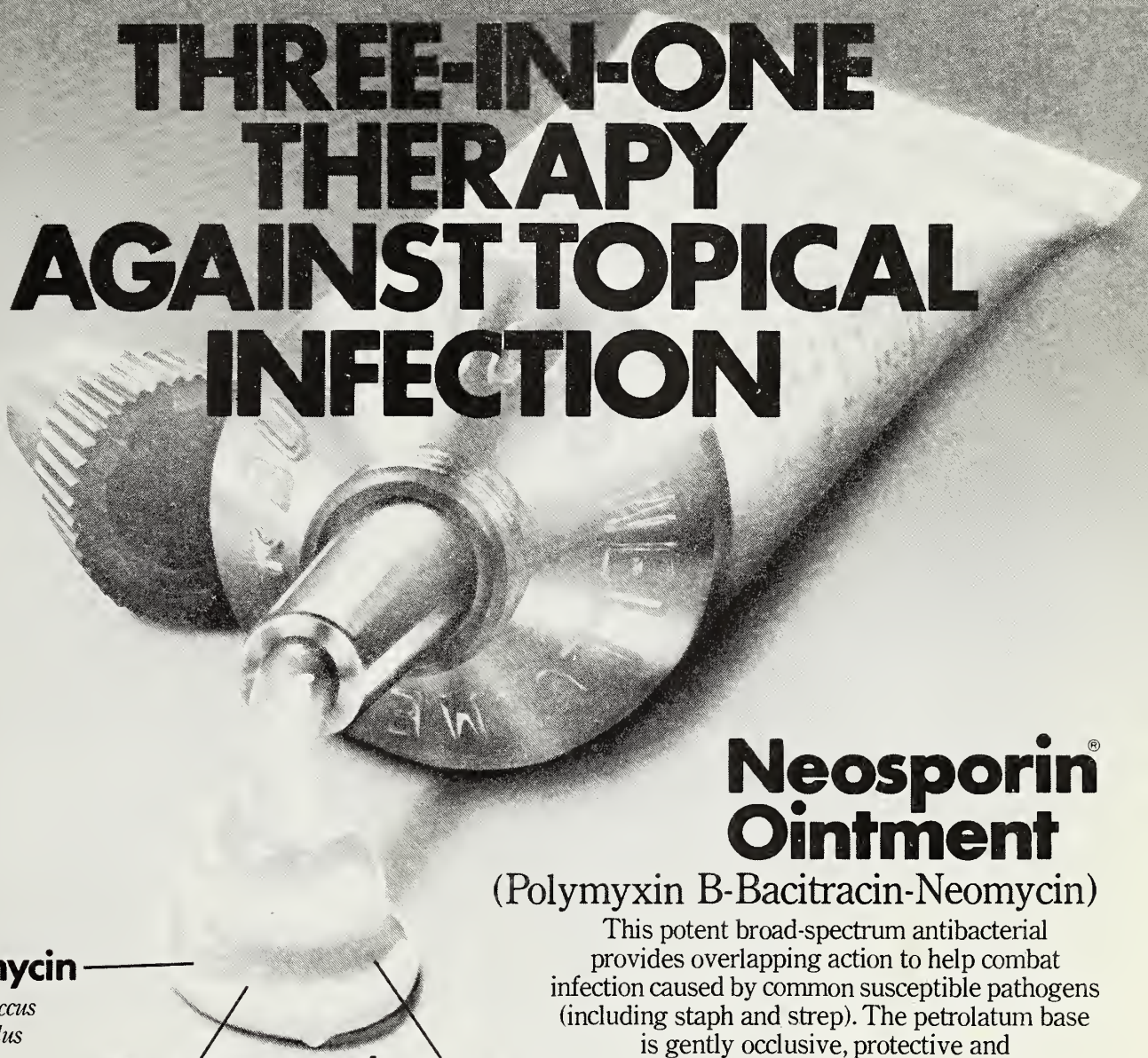
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All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

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Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

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Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated, avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment, blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, i.e., dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

ROCHE

Roche Products Inc.
Manati, Puerto Rico 00701



In treating certain G.I. disorders...

Enhance your therapeutic expectations with the triple benefits of

Adjunctive

Librax®

Each capsule contains
5 mg chlordiazepoxide HCl
and 2.5 mg clidinium Br.

antianxiety/antisecretory/antispasmodic

Librax is unique among G.I. medications in providing the specific antianxiety action of LIBRIUM® (chlordiazepoxide HCl) as well as the potent antisecretory and antispasmodic actions of QUARZAN® (clidinium Br) for adjunctive therapy of irritable bowel syndrome* and duodenal ulcer.*

ROCHE

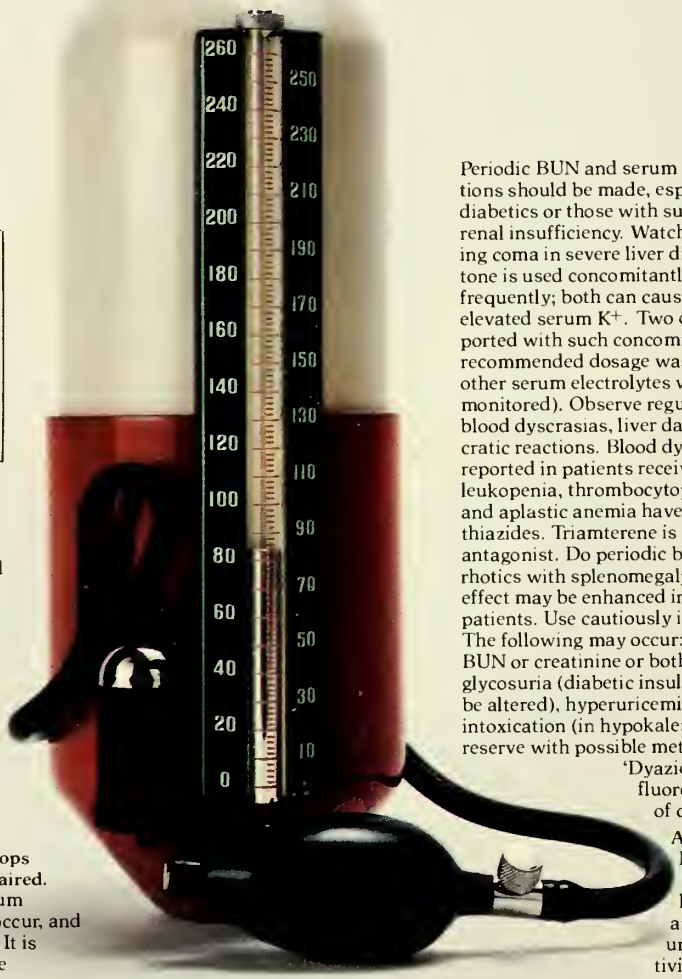
*Librax has been evaluated as possibly effective for this indication.
Please see brief summary of prescribing information on preceding page.

TRIAMTERENE CONSERVES POTASSIUM WHILE HYDROCHLOROTHIAZIDE LOWERS BLOOD PRESSURE

DYAZIDE®

Each capsule contains 50 mg. of Dyrenium® (triamterene, SK&F Co.) and 25 mg. of hydrochlorothiazide.

MAKES SENSE



Before prescribing, see complete prescribing information in SK&F Co. literature or PDR. A brief summary follows:

* **Warning**
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** When the combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome. Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium sparing action of triamterene is warranted. (See Box Warning.) Routine use of diuretics in healthy pregnant women is inappropriate; they are indicated in pregnancy only when edema is due to pathological causes.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

Supplied: Bottles of 100 and 1000 capsules; Single Unit Packages of 100 (intended for institutional use only).

FOR LONG-TERM CONTROL OF HYPERTENSION.* SERUM K^+ AND BUN SHOULD BE CHECKED PERIODICALLY. (SEE WARNINGS SECTION.)

SK&F CO., Carolina, P.R. 00630

SK&F CO.
a SmithKline company

ROCHE

For recurrent attacks of urinary tract infection in women

Bactrim™ DS Double Strength Tablets



Each tablet contains 160 mg trimethoprim and 800 mg sulfamethoxazole.

Just one tablet b.i.d. for 10 to 14 days

- Action at urinary/vaginal/lower bowel sites helps eliminate reservoirs of infecting organisms
- Distinctive antibacterial action plus wide spectrum helps eradicate recurrent UTI
- Low incidence of bacterial resistance in community practice
- Convenient *b.i.d.* dosage provides day-and-night antibacterial control
- Contraindicated during pregnancy and the nursing period. During therapy, maintain adequate fluid intake; perform CBC's and urinalyses with microscopic examination.

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* **It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination.** *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

The recommended quantitative disc susceptibility method (*Federal Register, 37*:20527-20529, 1972) may be used to estimate bacterial susceptibility to Bactrim. A laboratory report of "Susceptible to trimethoprim-sulfamethoxazole" indicates an infection likely to respond to Bactrim therapy. If infection is confined to the urine, "Intermediate susceptibility" also indicates a likely response. "Resistant" indicates that response is unlikely.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older:

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension,* containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

ROCHE

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

Please see back cover.

U. of California Medical Center
San Francisco, Calif. 94143

Her next attack of cystitis may require the Bactrim™ 3-system counteratta



ROCHE

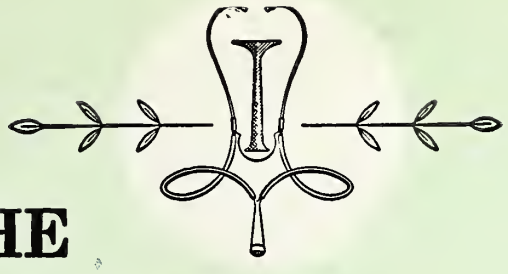
Bactrim has shown high clinical effectiveness in recurrent cystitis as a result of its wide spectrum and distinctive antimicrobial action in the urinary, vaginal and lower intestinal tracts.

The probability of recurrent urinary tract infection appears to be enhanced by the establishment of large numbers of *E. coli* or other urinary pathogens on the vaginal introitus. The trimethoprim component of Bactrim diffuses into vaginal fluid in effective concentrations, thus combating migration of pathogens into the urethra.

Studies have shown that Bactrim acts against *Enterobacteriaceae* in the bowel without the emergence of resistant organisms. Thus, Bactrim reduces the risk of introital colonization by fecal uropathogens. It has *no* significant effect on other normal, necessary intestinal flora.

Bactrim fights uropathogens in the urinary tract/vaginal tract/lower intestinal tract

Please see reverse side for summary of product information.



THE Journal of the Kansas Medical Society

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JULY
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The JOURNAL of the KANSAS MEDICAL SOCIETY

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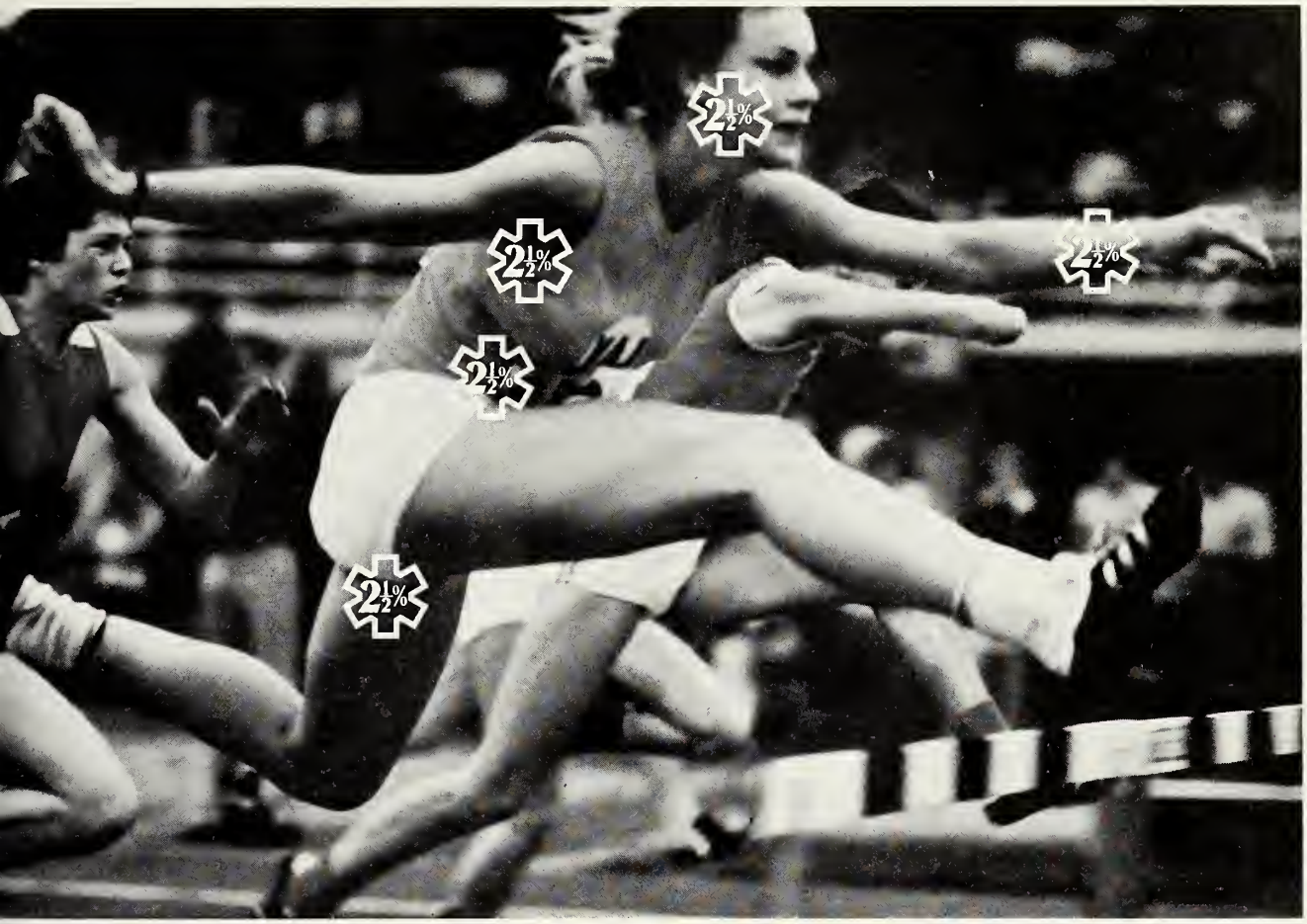
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Front Runner



HYTONE® 2½% Cream

(hydrocortisone)

for rapid control of the majority of inflammatory dermatoses seen in office practice—a logical alternative to the fluorinated steroids.

Followed by HYTONE® 1% or 1/2% Cream, for maintenance therapy and ultimate control.

Description: Hydrocortisone ½%, 1% or 2½% in a water-washable cream containing purified water, propylene glycol, glyceryl monostearate, cholesterol and related sterols, isopropyl myristate, polysorbate 60, cetyl alcohol, sorbitan monostearate, polyoxyl 40 stearate and sorbic acid

Action: Topical steroids are primarily effective because of their anti-inflammatory, antipruritic and vasoconstrictive actions

Indications: For relief of the inflammatory manifestations of corticosteroid-responsive dermatoses

Contraindications: Topical steroids are contraindicated in those patients with a history of hypersensitivity to any of the components of the preparation

Precautions: If irritation develops, the product should be discontinued and appropriate therapy instituted

In the presence of an infection the use of an appropriate antifungal or antibacterial agent should be instituted. If a favorable response does not occur promptly, the corticosteroid should be discontinued until the infection has been adequately controlled

If extensive areas are treated or if the occlusive technique is used there will be increased systemic absorption of the corticosteroid and suitable precautions should be taken, particularly in children and infants

Although topical steroids have not been reported to have an adverse effect on human pregnancy, the safety of their use in pregnant women has not absolutely been established. In laboratory animals, increases in incidence of fetal abnormalities have been associated with exposure of gestating females to topical corticosteroids, in some cases at rather low dosage levels. Therefore, drugs of this class should not be used extensively on pregnant patients, in large amounts, or for prolonged periods of time.

The product is not for ophthalmic use

Adverse Reactions: The following local adverse reactions have been reported with topical corticosteroids, especially under occlusive dressings: burning, itching, irritation, dryness, folliculitis, hypertrichosis, acneform eruptions, hypopigmentation, perioral dermatitis, allergic contact dermatitis, maceration of the skin, secondary infection, skin atrophy, striae, miliaria

Dosage and Administration: Apply to affected areas 3 or 4 times daily

How supplied: 2½% — Tube 1 oz., 1% and ½% — Tube 1 oz. and Jar 4 oz.

Caution: Federal law prohibits dispensing without prescription

For Patient Starters Call Toll-Free (800) 523-6674

Dermik

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

Librax®

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium Br.

Please consult complete prescribing information, a summary of which follows:

Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

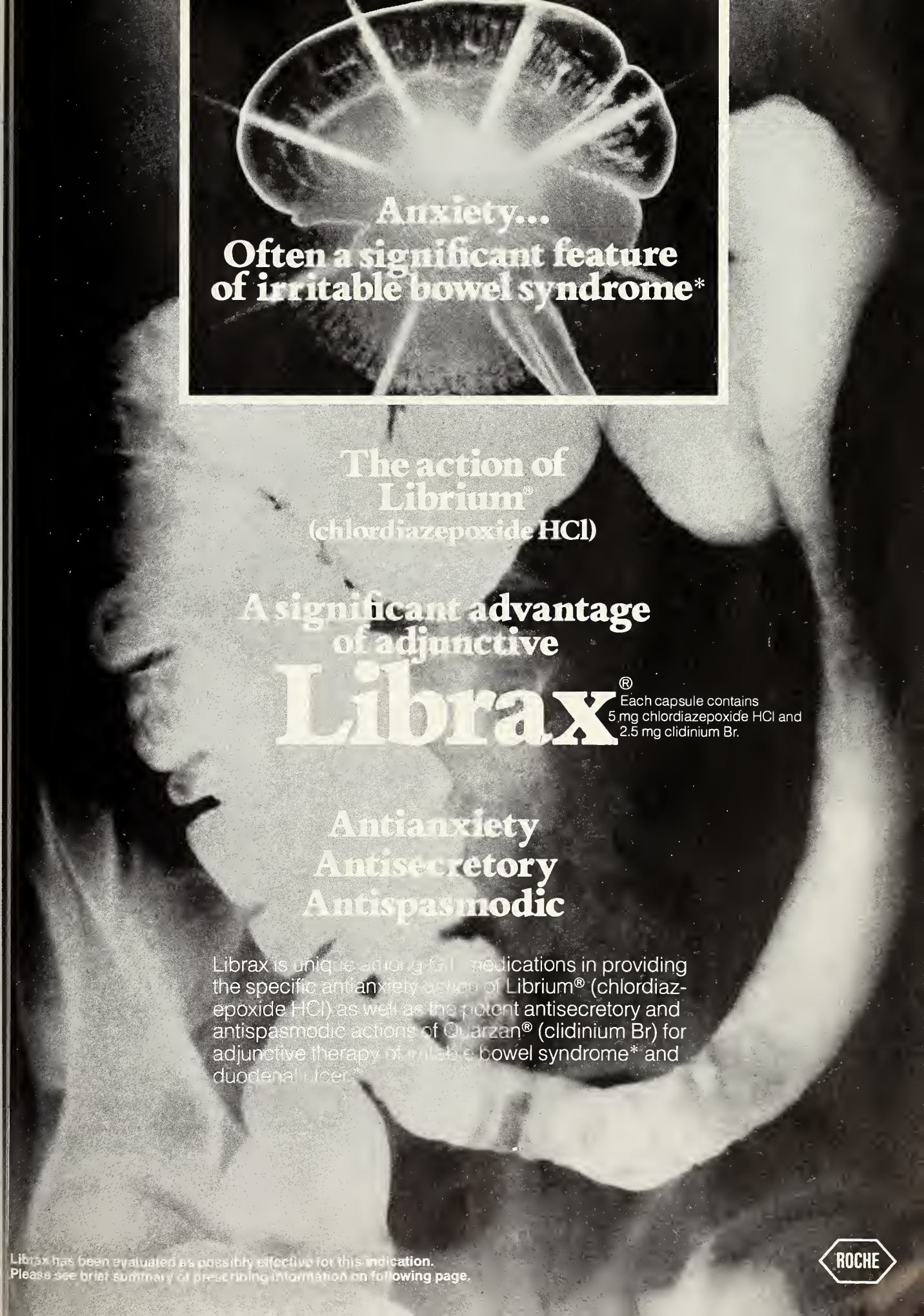
As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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Roche Products Inc.
Manati, Puerto Rico 00701


Anxiety...
Often a significant feature
of irritable bowel syndrome*
The action of
Librium®
(chlordiazepoxide HCl)
A significant advantage
of adjunctive
Librax®
Each capsule contains
5 mg chlordiazepoxide HCl and
2.5 mg clidinium Br.
Antianxiety
Antisecretory
Antispasmodic
Librax is unique among GI medications in providing
the specific antianxiety action of Librium® (chlordiaz-
epoxide HCl) as well as the potent antisecretory and
antispasmodic actions of Quarzan® (clidinium Br) for
adjunctive therapy of irritable bowel syndrome* and
duodenal ulcer.*
Librax has been evaluated as possibly effective for this indication.
Please see brief summary of prescribing information on following page.
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DRUG REGULATION REFORM ACT OF 1978

The Drug Regulation Reform Act of 1978, introduced into Congress on March 16, 1978, represents the first top-to-bottom overhaul of the Federal drug law since 1938, when the Food, Drug and Cosmetic Act was enacted. The eight major purposes of the reform act are:

To strengthen consumer protection

—by making it easier for the Food and Drug Administration to remove from the market an unsafe drug. (FDA now has to go through a lengthy administrative procedure to remove a drug from the market unless an "imminent hazard" is involved.)

—by confirming FDA's authority to require drug makers to monitor and report dangerous side effects after drugs are on the market. (FDA now has limited authority to require such information.)

—by establishing a single regulatory system for all human drugs, thus ending the current legal distinctions based on the type of drug or its date of marketing. (For example, there are separate systems for pre-1938 drugs, post-1938 drugs, antibiotics, insulin and vaccines, as well as for prescription and nonprescription drugs.)

To improve consumer information and understanding

—by requiring plain language information for patients about prescription drugs. (FDA now requires patient labeling for drugs such as birth control pills and estrogen used by menopausal women, but has no explicit mandate to expand this program.)

—by authorizing FDA to require informed consent by a patient before high-risk drugs are prescribed. (FDA now has no such authority.)

—by defining "safety" and "effectiveness" for new drugs in terms of a balancing of risks and benefits that the consumer can understand. (The bill would not change the present safety and effectiveness standard, merely define the terms more precisely.)

To encourage drug innovation

—by simplifying the current regulatory process: (See description below of how the system would work).

—by lowering industry costs of developing new drugs by reducing regulation in the early phases of drug research and by providing clearer guidance on what tests FDA will require.

—by providing better ground rules to help FDA and drug sponsors avoid and resolve disagreements during the testing stage of new drugs.

To make important new drugs available as rapidly as possible

—by allowing FDA to provisionally approve "breakthrough" drugs. (FDA has no authority now to permit early marketing of such drugs.)

—by allowing FDA to establish special distribution conditions for certain useful drugs that could not be approved if general marketing were the only option. (Now FDA has no such authority, and often must refuse to approve a drug for general marketing even when a small number of patients might benefit from it.)

—by allowing U. S. firms to export to other countries drugs not approved for use in the U. S. but which meet the regulatory rules and medical needs of the importing country. (FDA now has only narrow authority in this area.)

To promote greater competition among prescription drug suppliers

—by authorizing FDA to produce and distribute a drug compendium, a book for health professionals containing comprehensive information about drugs and their use. (FDA has no explicit direction now to publish such a compendium.)

—by requiring pharmacists to post drug prices, thereby making it easier for consumers to compare costs. (No such authority exists now.)

—by prohibiting gimmickry in drug promotion and by authorizing corrective advertising if advertising to professionals is misleading or inaccurate. (FDA now can prohibit false and misleading prescription drug advertising but cannot legally prohibit free samples or gifts to doctors designed to encourage them to prescribe a particular drug.)

To increase FDA openness and accountability

—by releasing to the public the safety and effectiveness data that provide the justification for FDA drug approvals. (FDA by law now must protect as "trade secrets" the data provided by industry.) Release of the data also would eliminate duplicative tests other companies must perform to demonstrate safety and effectiveness for the same chemical. This change would save scarce scientific resources, eliminate unnecessary testing of human subjects, and promote competition.

—by including the public in drug decisions through hearings, advisory committees, and government payment of attorney fees for citizen representation. (FDA now has consumer advisers, but there is no explicit provision

(Continued on page 306)

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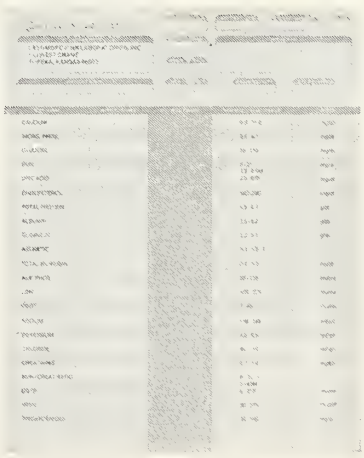
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Drug Regulation

(Continued from page 304)

in the new drug approval process for formal consumer contribution.)

To improve FDA's enforcement capability

—by giving FDA authority to embargo for up to 30 days shipment of a product it believes violates the law, while seizure processes are initiated in the courts. (Now, FDA's only options are to ask a manufacturer to withhold a product voluntarily or to ask state or local authorities to embargo it while FDA develops court papers to have it seized or enjoined.)

—by expanding FDA's inspectional authority and by giving FDA subpoena power.

—by allowing FDA to fine companies or individuals for serious, non-criminal violations of the drug law, after a hearing and subject to court review. (FDA has no authority now to impose such civil penalties.)

To focus attention on drug sciences and to encourage research and training

—by establishing a National Center for Clinical Pharmacology, which would conduct and support research and training in clinical pharmacology, study issues of drug regulation policy, and test drugs of little commercial value.

Why a New Law Is Needed

The 1938 Food, Drug and Cosmetic Act established the drug approval system now in effect. This law was passed after a drug disaster killed more than 100 persons in 1937 (the "elixir of sulfanilimide" episode in which the drug was tested for flavor, appearance and fragrance, but not for safety). There have been several drug amendments since 1938. The most important were in 1962, when the requirement was added that drugs had to be proved not only safe but effective as well. The 1962 amendments also grew out of a disaster—the sedative thalidomide, never approved in the U. S., caused serious birth defects overseas.

Since 1938, there have been dramatic changes in the use of drugs. Some 60,000 prescription and 200,000 to 300,000 nonprescription drugs are now available. Ninety per cent of all prescriptions today are for drugs that were not available in 1938. In 1964, doctors wrote 860 million prescriptions; in 1977, they wrote 1.5 billion, an average of 5.7 for every American. Americans spend $16 billion a year on prescription and nonprescription drugs.

On October 5, 1977, Secretary Joseph A. Califano, Jr., the Department of Health, Education and Welfare, announced that comprehensive legislation was needed to restructure the present regulatory system. The drafting process then began, and in November, FDA held public hearings to receive comment on what should be in the legislation. Extensive discussions also were held with Congressional staffs. The bill thus incorporates public thinking as well as industry and professional group contribution.

How the Drug Review System Would Work

Under the proposed new law, the drug research phase would be divided into two phases: an "innovational" or discovery phase, and a "developmental" stage. FDA would be less concerned with the scientific design and results of the investigations during the innovational stage, thus reducing regulation of this stage of drug research without compromising the safety or rights of human subjects.

When the investigations have been completed, the drug sponsor would file two documents with FDA: a petition to establish a "monograph" (a statement of what the drug is, what it does, conditions for dispensing, required labeling, etc.), and an application to market the drug under that monograph.

At the time of filing, summary information on the drug's safety and effectiveness would be made available to the public. During the review process, the data submitted by the drug sponsor would be made available to the public on a restricted basis (to prevent exploitation by competitors), and FDA would hold a public hearing. (Currently, brief summaries of the data are released after the approval decision is made, and no public hearing is required.)

Petitions on important new drugs and questions on significant regulatory decisions would be submitted to an advisory committee, consisting of nongovernment experts, for its recommendation. This will assure further public involvement in the decision-making process.

Once FDA is assured that the drug is safe, effective and properly labeled, the Agency would approve the monograph as well as the company's license to market the drug. FDA could require a patient package insert for the drug so consumers would understand its benefits, its risks, and how to use it most effectively and safely.

After a monograph is established, other manufacturers who want to market the same drug need only apply for a license under the monograph (consistent with patent laws and other provisions of the drug law). The bill permits other manufacturers to rely on data developed by the original manufacturer, but only after five years have elapsed since approval.

(Continued on page 344)

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Auxiliary News

At the present time, plans are being made for the KMS Auxiliary Summer Board Meeting to be held in Topeka, July 13-14. A business meeting on Thursday afternoon will include reports from some of the major committees. Membership has always been, and will continue to be, a primary concern for us, since our effectiveness depends on a large and concerned membership. Please urge the spouse in your house to join us—we can do more together. The year ahead will be another crucial year on legislative issues, and we stand ready to assist you in any way you may deem advisable.

Friday morning, July 14, will be devoted to workshops. These workshops are designed to assist County Auxiliaries as they plan and implement community projects. Since our emphasis this year is on Health Education for Children and Youth, two of the workshops will cover the Learning Center on Body Pollution and the new program on Health Education for teenagers. A third workshop will instruct new chairmen in our fund raising efforts via AMAERF. The fourth workshop will concern the other new program the Auxiliary has adopted this year, the Liaison to the KMS Committee on the Impaired Physician Program.

We sincerely hope that as physicians' spouses continue to work in their own communities an awareness of our concern for the well-being of all people will be apparent to members of the community.

By the time you read this, the AMAA convention will be history and I will report on its highlights in a later issue.

An added note: One of the actions of the Board at our state convention was the allotment of $500 to the International Health Activities Committee, to enable Mrs. W. G. Cauble, Wichita, Chairman, to move IHA material. One of the major problems of this committee has been the shipping of drugs, medical supplies, medical journals, etc. once they have been collected. Dee is one of the most dedicated gals I know and deserves our wholehearted support and applause for her activities.

From all the members of the KMS Auxiliary—thanks for your interest, your support, and your dedication to your profession. Though we may sometimes fail to tell you, individually, we are proud of the "spouse in our house," our doctor, and we stand ready to serve you. Don't fail to ask us. We're probably the best "cheering section" you have.

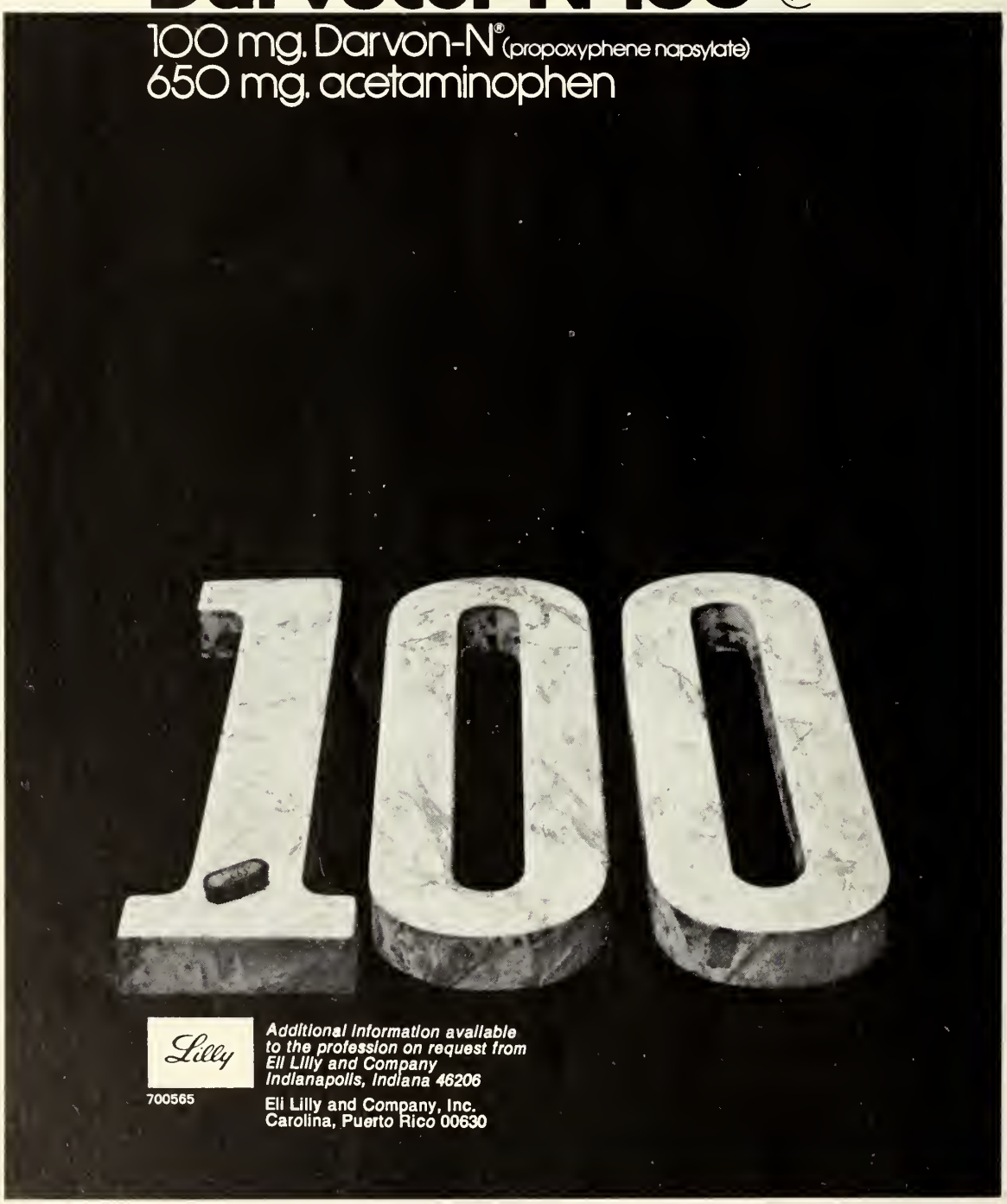
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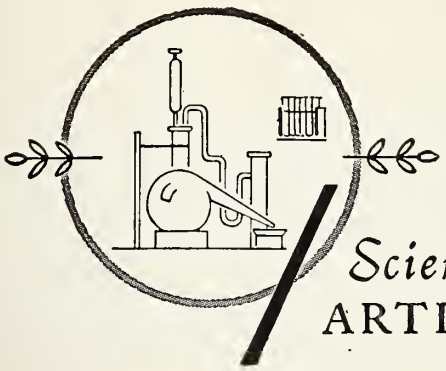


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Scientific ARTICLES

Immunodeficiency

Detection of Immune Deficiency States by the Primary Care Physician

DAVID L. STRAUB, M.D.,
ARNALDO CAPRILES HULETT, M.D., *and*
ROBERT J. DOCKHORN, M.D., *Kansas City*

IMMUNE DEFICIENCY STATES have been regarded as rare, "medical center" diagnoses. Their detection appears to be increasing in the past few years, however. We believe this is due to increased awareness. The following discussion demonstrates an approach utilized by primary physicians to detect deficiencies in the immune system by employing tools that are readily available.[1-4] It is emphasized that immune screening can be done at the local primary care level. Examples of immune deficiency states are presented to illustrate this point.

With the accumulation of knowledge, the complexity of the body's immune system has become apparent. Although its interrelating functional components should not be oversimplified, it is appropriate to systematically evaluate the B-cell (humoral), T-cell (thymic dependent), phagocytic, and complement properties. The following discussion is not meant to be a detailed analysis of immunodeficiency, but hopefully will be a useful guide for the practitioner.

The *B-cell system* provides secretory and circulating antibodies to prevent and fight bacterial and viral infections, parasitic infestations, and some malignancies. The *T-cell lymphocytes* augment or suppress antibody function. They are involved in delayed hypersensitivity, graft-versus-host reactions, transplantation reactions, malignancy surveillance, and defense against viral and fungal infections. The *phagocytic cells* intercept and process antigens and aid in the killing and clearance of bacteria. The *complement system* is a series of glycoproteins that act as mediators in producing chemotaxis, anaphylaxis, and cell lysis. The B- and T-cell defects comprise by far the greatest number of immunological problems.[5, 6] The following paragraphs outline a systematic approach to their evaluation.

Previously rare immunodeficiency states are increasingly being reported because of increased awareness. The great majority can be diagnosed by the primary care physician utilizing basic knowledge of the immune system and tools that are readily available. Rapidly advancing knowledge that is providing treatment for immune defects makes it imperative that physicians consider and screen for immunodeficiency appropriately.

History[7]

As with any medical problem, an accurate history is

Address reprint requests to Dr. Dockhorn, 5300 West 94th Street Terrace, Prairie Village, KS 66207.

the first step. A familial history of immune deficiency, unusual or severe infections, collagen disorders, and early infant death may signify an immunity problem. Parental consanguinity increases the chance for expression of an autosomal recessive problem. Other immunodeficiencies are x-linked and found only in males.

A personal history of infections of unusual type, unexpected severity, or increased frequency is very important. Some indicators of immunodeficiency are: (1) a severe clinical course or unusual complication from a relatively minor illness; (2) infections by Serratia marcescens, Pneumocystis carinii,[8] or other unusual pathogen; (3) a history of recurrent pneumonias, persistent sinusitis, or bronchiectasis; (4) prolonged or recurrent diarrhea; (5) failure to thrive; and (6) serious complications from administration of live virus vaccines.[9, 10]

The age of onset and clinical association of the patient's symptoms may also provide a clue. Severe combined immunodeficiency (B-cell and T-cell) is manifested by the occurrence of infections in the first few weeks of infancy. However, recognition of pure B-cell or humoral defects could be delayed until six months of age or longer because of protection by placentally-transferred maternal IgG antibodies. Tetany in the newborn may be a manifestation of the DiGeorge syndrome with its T-cell defect. Dysgammaglobulinemia with elevated IgM has occurred in patients with congenital rubella. Rarely, infectious mononucleosis is followed by hypogammaglobulinemia.[5, 6]

Signs that may demonstrate immunity competence include normal growth and development and complete recovery following viral and bacterial infections. Rhus dermatitis and the expected appearance and resolution of an exanthem with viral infections suggest intact T-cell immunity.[11] It is important to remember that the normal child may have 6-12 upper respiratory infections yearly and may be well between them. The immune deficient patient tends to present a continuum of illness.

Physical Examination

The physical examination of an immunity deficient patient may be entirely normal. However, children may appear chronically ill, fail to thrive, and exhibit mental and motor development delays. Evidence of recurrent or persistent infection should be sought especially in the respiratory system. Bronchiectasis, otitis media, and sinusitis are common complications. Examination of lymphoid tissue may reveal hyperplasia or hypoplasia. In addition to evidence of infection, other clues are shown for representative syndromes in *Table 1*. More sophisticated investigations, such as T- and B-cell studies, that are not available in most local laboratories are not included.

Laboratory

Laboratory investigation should be initiated in a systematic manner, including both quantitative and functional aspects of the immunological components. Although the following laboratory determinations are categorized, some deficiencies will involve more than one division. It is important to differentiate whether a particular deficiency is primary or secondary to another disease process, such as loss of immunoglobulin in the urine of children with nephrotic syndrome.

B-cell System

Evaluation of humoral or B-cell immunity[1, 4, 12-14] begins with the demonstration that all of its elements are present in normal amounts. In addition to physical examination of lymphoid organs, a lateral x-ray of the neck can show the presence or absence of an adenoidal shadow. Quantitation of the immunoglobulin levels of serum IgG, IgA, and IgM is easily done by radial immunodiffusion using Mancini plates that are readily available commercially. These studies may also be done on body secretions such as saliva or tears, if a secretory antibody deficiency is suspected. Radial immunodiffusion is more accurate than the older method of immunoelectrophoresis.

The intact function of the humoral system can be shown by measuring specific antibody titers. IgM response is demonstrated by the presence of isohemagglutinin titers of 1:4-1:8 or greater after six months of age. Although the Schick test is no longer readily available, IgG response can be shown by the presence of antistreptolysin (ASO), polio, rubella, diphtheria, and tetanus titers if the patient has contracted these organisms or received immunizations against them. ASO and rubella titers are usually available locally, while state laboratories provide measurement of the diphtheria and tetanus levels. One way of demonstrating both IgG and IgM functions is by administering three weekly injections of typhoid vaccine 0.5 ml, and then obtaining the febrile agglutinins to H antigen (IgG response) and O antigen (IgM response). However, caution should be used, and no such patient should receive live or attenuated virus immunizations.[9, 10]

Interpretation of tests of B-cell anatomy and function depends on age, possible presence of transplacentally acquired IgG, and prior gamma globulin treatment.

T-Cell System

The anatomy of the T-cell (thymic dependent) sys-

TABLE I

PHYSICAL AND LABORATORY FINDINGS IN IMMUNODEFICIENCY

Syndrome	*Clinical Features*	*Laboratory Findings**
Transient hypogammaglobulinemia of infancy	Normal	Low IgG, IgM, IgA, IgD
X-linked hypogammaglobulinemia	Male; small or absent lymph nodes, tonsils, and adenoids; malabsorption; arthritis.	Low IgG, IgM, IgA, IgD
X-linked hypogammaglobulinemia with normal or increased IgM	Male; normal or enlarged lymph nodes, tonsils and adenoids; increased incidence of B-cell lymphomas.	Low IgG, IgA; normal or increased IgM; neutropenia, thrombocytopenia, hemolytic anemia.
Acquired hypogammaglobulinemia (common variable immunodeficiency)	Onset in late childhood or adulthood; lymph nodes of variable size; increased incidence of pernicious anemia and lymphoreticular malignancies.	Low IgG, IgM, IgA, IgD
Deficiency of IgA	Increased incidence of respiratory infections, allergy, celiac disease, rheumatoid arthritis, and systemic lupus erythematosus.	Low IgA in serum and secretions; normal or elevated IgG, IgM, IgD.
Severe combined immunodeficiency	X-linked or autosomal recessive; infections in early infancy; diarrhea; failure-to-thrive; absent lymph nodes, tonsils, and adenoids.	Lymphopenia; negative delayed hypersensitivity skin tests; absent thymic and adenoidal shadows on x-ray; low IgG, IgM, IgA, IgD.
Congenital thymic aplasia (DiGeorge syndrome)	Neonatal tetany; congenital heart disease (aortic arch abnormalities); hypoparathyroidism; hypertelorism; notched ear pinnae; micrognathia; low set ears; antimongoloid slant of eyes; nephrocalcinosis.	Normal or low absolute lymphocyte count; hypocalcemia; elevated phosphorus; absent thymic shadow on x-ray; negative delayed hypersensitivity skin tests.
Wiskott-Aldrich syndrome	Male; eczema; failure-to-thrive; petechiae; bleeding tendencies; increased incidence of lymphorecticular malignancies.	Thrombocytopenia; lymphopenia; Coomb's positive anemia; normal IgG; low IgM; increased IgA, IgD, IgE; absent isohemagglutinins; negative delayed hypersensitivity skin tests.
Chronic mucocutaneous candidiasis	Chronic candida infection; hypoparathyroidism; Addison's disease; hypothyroidism; pernicious anemia; gonadal dysgenesis.	Negative delayed hypersensitivity skin test to candida antigen; other delayed hypersensitivity skin tests positive.
Ataxia-Telangiectasia	Ataxia; telangiectasia on bulbar conjunctiva and skin; sinopulmonary tract infections; increased incidence of lymphosarcoma.	Normal or low IgA and IgE; lymphopenia; absent delayed hypersensitivity skin tests.
Chronic granulomatous disease	Abscess formation of any organ; enlarged lymph nodes, liver and spleen; eczematoid or seborrheic dermatitis; diarrhea.	Abnormal nitroblue tetrazolium dye test; leukocytosis; anemia; elevated IgG, IgM, and IgA.

* Only abnormalities which can be detected in the local laboratory are included.

TABLE II
SCREENING PROCEDURES FOR IMMUNODEFICIENCY

Humoral (B-cell)
- Lateral x-ray of neck for adenoidal shadow
- IgG, IgA, IgM, IgD
- Antibody titers: isohemagglutinins, antistreptolysins, rubella, diphtheria, tetanus, polio, typhoid

Thymic Dependent (T-cell)
- Chest x-ray for thymic shadow
- Absolute lymphocyte count
- Peripheral blood smear
- Skin tests for delayed hypersensitivity: candida, fluid tetanus toxoid, tuberculin, histoplasmin, mumps, trichophyton, streptokinase-streptodornase

Phagocytosis and Complement
- Total white cell count and differential
- Total hemolytic complement
- Quantitative complement components

tem[1-4, 15, 16] is established by a normal absolute lymphocyte count (per cent of lymphocytes multiplied by total WBC) and normal, small lymphocytes on a peripheral blood smear. Because approximately 70 per cent of circulating lymphocytes are T-cells, an absolute lymphocyte count of less than 1200 cells/mm^3 may indicate T-cell deficiency. The presence of a thymic shadow is variable, but should be found in a patient under 12 mos of age if he is not in distress or has received steroid treatment.

There are several skin tests to detect the delayed hypersensitivity function of the T-cell division. These include the tuberculin, histoplasmin, mumps, streptokinase-streptodornase, trichophyton, candida, and fluid tetanus toxoid skin tests. If the patient has received tetanus immunization, fluid tetanus toxoid and candida may be the most useful antigens. A 10-mm or greater area of induration 48-72 hrs after intradermal injection of antigen 0.1 ml is considered positive. The fluid tetanus toxoid is obtained from the tubes used for tetanus immunization. Candida skin testing is started at the dilution of 1:1000. If negative, concentration of 1:100 is used. Because of possible severe tissue reactions, we do not advocate routine screening by topical application and subsequent challenge with dinitrochlorobenzene.

Phagocytic and Complement Systems

Phagocytic and complement screening[17-21] at the local level may be difficult. Fortunately, these defects are more rare than B- or T-cell problems, but investigation is required if answers are not found in the latter two systems. Normal phagocytic function may be screened by obtaining a complete blood count and absolute polymorphonuclear count. The nitroblue tetrazolium test (NBT) has been found useful in detecting chronic granulomatous disease and its carrier state.[22] Quantitative levels for complement components and total hemolytic complement can be screened using radial immunodiffusion on Mancini plates. The only factor against their use in a smaller hospital is the rarity of need.

Case Reports

Case One

An 8-day-old male was admitted for evaluation because an older brother had died at the age of four months with a diagnosis of thymic alymphoplasia. The parents are first cousins, and the mother has had at least three early spontaneous abortions. Physical examination was normal except that no lymphoid tissue could be found. The laboratory data revealed the following findings: white blood cell count (WBC), 3,700/cu mm with 49% segs, 12% bands, 18% lymphocytes, 5% monocytes, and 16% eosinophils. The total lymphocyte count was 666 cells/mm^3; IgG, 1,600 mg/dl; IgA, 0 mg/dl; IgM, 7.6 mg/dl; IgE, 1 unit/ml; T-cell rosettes, 0; adenosine deaminase, 0. Adenosine deaminase in the mother and father were 28 and 29 uM/hr/gm/Hb respectively. No thymus could be seen on x-ray. The diagnosis of severe, combined immunodeficiency was confirmed. The elevated IgG was due to transplacentally acquired maternal IgG.

COMMENT

This patient was identified because his referring physician had a high index of suspicion. Important points to consider are sex (male), history of abortions, consanguinity, sibling history of immunodeficiency, absent lymphoid tissue, absent thymic shadow, low total WBC, and decreased absolute lymphocyte count. The other studies were confirmatory.

Case Two

A 17-month-old male presented with a history of failure to thrive, recurrent colds, draining ears, and a diffuse eczematoid rash since birth. No lymphoid tissue could be found on examination. The following laboratory data were obtained: WBC, 1,200/cu mm with 51% segs, 35% lymphocytes, 4% monocytes, and 10% eosinophils; platelets, 10,000/cu mm; absolute lymphocyte count, 4,480 cells/mm^3; T-cell rosettes, 10%; lymphocyte PHA stimulation, negative; candida and SK-SD skin tests, negative; isohemagglutinins, negative; blood group, O; IgM, 43 mg/dl; IgA, 410 mg/dl, IgG, 588 mg/dl; IgE, > 1,400 units/ml.

COMMENTS

The patient's male sex, failure to thrive, eczema, recurrent ear infections, thrombocytopenia, and evidence of T- and B-cell dysfunction point to a diagnosis of Wiskott-Aldrich syndrome.[23]

Case Three

A 12-year-old male presented with a four-year history of cough and recurrent infections of the middle ears, maxillary sinuses, and lower respiratory tract. Immunoglobulin assay by his referring physician indicated low IgG, IgM, and IgA levels. Rales were audible in the left side of the chest posterior. X-ray showed the presence of pneumonia in the left-lower lobe and clouding both maxillary sinuses. Laboratory studies revealed the following values: IgG, 42 mg/dl; IgA, 3.8 mg/dl; IgM, 4.5 mg/dl; IgE, <10 units/ml; isohemagglutinins, weakly positive; RAST, negative; WBC, 12,800/cu mm with 72% polycytes, 5% Stabs, 19% lymphocytes, 3% monocytes, 1% eosinophils; absolute lymphocyte count, 2,432/cu mm^3; platelet count, 283,000/cu mm; total hemolytic complement, 250 C'H 100; C'3, 245 mg/dl; C'4, 67 mg/dl; C'5, 10.5 mg/dl; T-cell rosettes, 43%; B-cell markers: IgG, 9%, IgA, 1%, IgM, 10%, IgE, 1%; nitroblue tetrazolium test (NBT), 93%; sweat test, 15 mEq/dl; intradermal skin test to candida albicans, positive.

COMMENTS

Our patient's recurrent sinopulmonary infections led us to evaluate his immunoglobulin levels, an investigation that helped establish a diagnosis of acquired hypogammaglobulinemia (common variable immunodeficiency).[24] Results of laboratory evaluation of the other immune parameters were normal.

Case Four

A 4-month-old female infant was admitted following a month of cold symptoms and subsequent development of diarrhea and dehydration. All parameters of growth were below the third percentile. Examination revealed oral thrush and bilateral pneumonia. *Escherichia coli* was cultured from the blood and urine. Laboratory data were as follows: WBC, 11,400/cu mm with 77% segs, 15% bands, 2% lymphocytes, 6% monocytes; absolute lymphocyte count, 228/cu mm^3; T-cell rosettes, 1%; candida skin test, negative; IgG, 92 mg/dl; IgA, 9.5 mg/dl; IgM, 30 mg/dl; IgE, 5 units/ml; NBT, 92%; C'3, 135 mg/dl; sweat test, 27 mEq/dl; chest x-ray showed no thymic shadow.

COMMENTS

The patient's history of prolonged respiratory symptoms, diarrhea, failure to thrive, oral thrush, and sepsis are secondary to a severe combined immunodeficiency that was demonstrated by the above abnormal T- and B-cell indicators. Lymphopenia was a prominent sign of T-cell dysfunction, and prompted the search for an immune problem.

Case Five

An 8-month-old male presented with a history of prolonged diarrhea (>2 mos), generalized monilial rash in early infancy, and chronic cough. Tonsillar tissue was present and the physical examination produced normal values. Laboratory data were as follows: WBC, 8,800/cu mm with 74% segs, 2% bands, 15% lymphocytes, 8% monocytes, 1% eosinophils; absolute lymphocyte count, 1,320/cu mm^3; T-cell rosettes, 12%; skin tests to candida albicans and fluid tetanus toxoid, negative; IgG, 0 mg/dl; IgA, 4 mg/dl; IgM, 54 mg/dl; IgE, 5 units/ml; NBT, 95%; total hemolytic complement, 140 C' H100; C'3, 140 mg/dl; B-cell markers: IgA, 15%; IgG, 30%; IgM, 27%; total B-cells, 72%.

COMMENTS

Even though the results of the physical examination were normal, the history of prolonged diarrhea, generalized candidiasis, chronic cough, low normal lymphocyte count, negative delayed hypersensitivity skin tests, and low immunoglobulin levels are all clues to the diagnosis of severe combined immunodeficiency.

Case Six

A 10-month-old male was admitted to the hospital with a four-month history of recurrent respiratory tract infection. Twelve courses of antibiotics had been administered. Physical examination revealed a toxic-appearing child with a weight at the 25th percentile. His hospitalization was stormy, with diarrhea, pneumonia, marked abdominal distension, high fever, and pronounced weight loss requiring total parenteral alimentation. During this time, he also developed osteomyelitis involving the fourth finger of the left hand and several cutaneous abcesses that grew Pseudomonas cepacia. Laboratory data were as follows: WBC, 20,400/cu mm with 59% segs, 12% bands, 28% lymphocytes, 1% monocytes; IgG, 2,452 mg/dl; IgA, 318 mg/dl; IgM, 138 mg/dl; IgE, 90 units/ml; RAST, negative; isohemagglutinins, positive; T-cell rosettes, 25%; absolute lymphocyte count, 5,712/cu mm^3; candida and tetanus toxoid skin test, positive. Gastrointestinal roentgenogram showed changes compatible with lymphoid hyperplasia of the jejunum; NBT, 0% (no dye reduction).

COMMENTS

This case illustrates that when history and clinical

course strongly indicate the possibility of an immune deficiency, investigation should be pursued even if previous B-cell and T-cell screening methods are not informative. The negative dye reduction shown by the nitroblue tetrazolium test confirmed the diagnosis of chronic granulomatous disease.

Case Seven

A 33-month-old male presented with a history of failure to thrive, chronic diarrhea, and persistent otitis since early infancy. Height and weight were below the third percentile. Purulent drainage came from the nose and both auditory canals. Laboratory data were as follows: WBC, 6,100/cu mm with 7% segs, 12% bands, 75% lymphocytes, 2% monocytes, and 4% eosinophils; platelet count, 395,000/cu mm; absolute polycytes, 1,159/cu mm; absolute lymphocytes, 4,575/cu mm; hemoglobin, 10.3 gm/100 ml; hematocrit, 30.7%; sweat chloride, 9 mEq/dl; IgM, 209 mg/dl; IgE, < 5 units/ml; IgD, 4.1 mg/dl; B-cell markers: IgG, 16%, IgA, 2%, IgM, 19%, isohemagglutinins, negative; secretory IgA in tears, 5.5 mg/dl; T-cell rosettes, 7%; delayed skin tests with fluid tetanus toxoid and candida (1/1,000 and 1/100), all negative; chest x-ray, left-lower lobe infiltrate, no thymic shadow; total complement, C'3, C'4, NBT, normal.

COMMENTS

The data indicate T- and B-cell defects. It is remarkable that with such significant clinical problems, severe immune defects, and almost no growth in the preceding 12 months he escaped detection until 33 months of age. He had received two live polio vaccines without problems.

Summary

This paper demonstrates that diagnosis of immunological problems can be made employing history, physical examination, and commonly available laboratory procedures. Clinical examples illustrate this point. Other clinical entities such as cystic fibrosis, allergy and anatomical defects are important, but immune deficiency should be included in the differential diagnosis of patients with recurrent infection.

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Clinical Electromyography

When and How to Use It

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ELECTROMYOGRAPHY (EMG) is a relatively new technique of studying disorders of the nerves and muscles. During the past ten years, great advances have been made in the diagnosis and treatment of musculoskeletal and neuromuscular disorders by electrical means, but most physicians are only superficially acquainted with these advances. The purpose of this paper is to outline the clinical applications of EMG and give some guidance to conditions that might be referred for testing using this technique. Although only those physicians fully acquainted with disorders of the nervous or muscular apparatus are capable of applying or interpreting electromyography, most physicians should be familiar with its usefulness.

Galvani may be considered the originator of electromyography. As early as 1794, he described the existence of electricity resulting from muscular contraction. Nobili (1827), Mateucci (1838), Dubois-Reymond (1851) and others have all contributed to the early work in clinical electromyography, but the apparatus in use was too insensitive for clinical investigation. Muscle electricity remained a scientific curiousity until the arrival of vacuum tube amplifiers and cathode ray oscilloscopes in the third decade of the 20th century. The credit for launching modern electromyography goes to Adrian and Bronk, who developed a concentric needle electrode in 1929. Since then, significant advances have been made in equipment and clinical use of electromyography, so that recording potentials from sensory nerves in the order of 5-10 microvolts has become a common practice.

There are three basic areas in which EMG is used today: (1) clinical evaluation, diagnosis, prognosis, and treatment in disorders of the neuromuscular system; (2) research studies in physiology and neuropathology; and (3) kinesiology and its practical applications. Only the clinical application of EMG will be discussed here; its newer applications, such as biofeedback, will not be considered.

Basically, EMG can be applied to any lesion involving the anterior horn cells, axons, neuromuscular junction, and skeletal muscles as these generally result in abnormal electromyographic patterns. On the other hand, electromyography, as generally conducted, is limited in value in differential diagnosis of upper motor neuron lesions. However, negative EMG finding in a patient with muscle weakness may be useful as suggestive of a central nervous system disorder. There are two parts to clinical electromyography:

1. Needle electromyography in which the integrity of the motor unit is studied by needles directly in the muscles;

2. Evoked potential electromyography—or more correctly electroneurography—in which the nerve is stimulated and a response recorded from the muscle or from the sensory nerves.

The technique of clinical electromyography is a useful extension of the neurologic examination for diagnosing and prognosing disorders of the neuromuscular system. The process is explained and its various applications are discussed and evaluated.

Needle Electromyography

Needle electromyography is a study of a minute area of skeletal muscle to record electrical activity by sampling a particular area of skeletal muscle, generally using 25-gauge teflon coated needles. As the signals are extremely weak, it is necessary to amplify the impulses considerably before they can be displayed. Needle electrodes connect to a special type of amplifier to magnify the miniscule electromyographic signals while rejecting such sources of interference as alternating line current. The amplifier output is displayed on a cathode ray oscilloscope for visual interpretation and on a loudspeaker for aural interpretation. Permanent records may be made by photographing the oscilloscope tracings or by using magnetic tapes or light sensitive paper. *Figure 1* shows the usual components of an electromyographic recording system, with which the electrical activity of the motor unit or the basic unit of contraction in muscles

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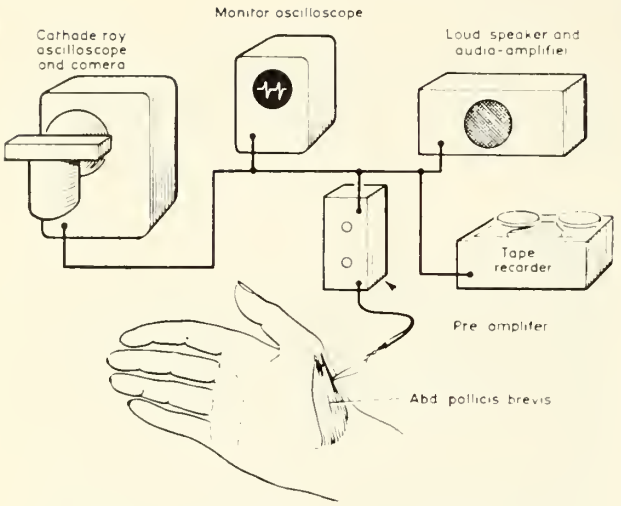


Figure 1. Diagram of general arrangement of monitoring and recording apparatus (after Basmajian).

is recorded *(Figure 2)*. The recording from one motor unit is called a motor unit action potential. Only a very limited number of motor units can be recorded for any muscle sampled.

Electromyography has been compared to the determination of defects in a central telephone system, by studying output at the subscribers' end of the telephone. When considering such an analogy, the limitations and technical problems immediately become apparent; it is a sampling with many subjective features, not a completely objective test. With a needle electrode inserted in a normal muscle at rest, no electrical activity is recorded. Upon minimal voluntary contraction, rather simple wave forms—usually biphasic or triphasic—start appearing in rhythmic patterns of 5-10/second. As the muscle is contracted more and more strongly, frequency of occurrence of the individual motor unit action potentials increases and new potentials appear. This increase in frequency and number is called recruitment. At maximal contraction, it is no longer possible to distinguish individual potentials, and the resulting jumbled appearance is called an interference pattern. With complete loss of function of lower motor neuron, the obvious clinical change is the inability to contract the muscle voluntarily and no voluntary motor unit action potentials appear on electromyography. This is termed electrical silence. However, when Wallerian degeneration has been completed (at the end of 2-3 weeks) muscle fibers contract individually and independently, producing a phenomenon known as fibrillation and recorded as minute, irregularly occurring waves in electromyography called fibrillation potentials. In the usual clinical situation with moderate numbers of fibrillation potentials present, the sound is

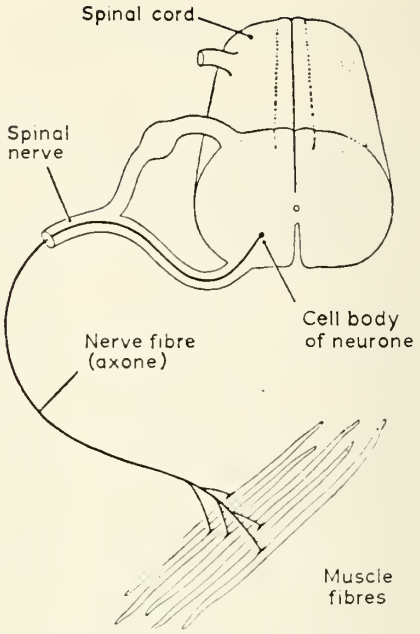


Figure 2. Diagram of a motor unit (after Basmajian).

like the crackling of tissue paper; but in acute, severe, lower motor unit disorders, fibrillations may be diffuse, and resulting noise from the loudspeaker is described as similar to hail on a tin roof. In muscles where the membrane is unstable, potentials having a distinctive V shape are seen in addition to fibrillation potentials. These are called positive sharp waves. These positive sharp waves apparently originate from the injured muscle fiber and are seen earlier after injury than fibrillation potentials, often at the end of one week. Fibrillation potentials are due to increased sensitivity of the denervated muscles to small quantities of acetylcholine which stimulate muscle contraction at sites removed from the neuromuscular junction. (*Figure 3* shows fibrillations and positive sharp waves.)

After studying the muscle with the needle electrode at rest, the clinical electromyographer asks the subject

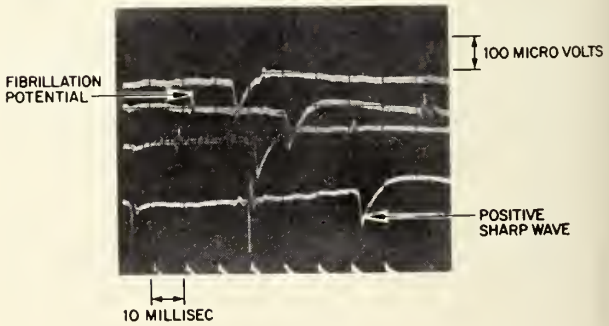


Figure 3

to contract the muscle. If the lower motor neuron involvement is only partial, some motor unit action potentials will appear. However, certain changes may be noted in motor units in partial lower motor neuron involvement. Voltages of potentials may be higher than normal, their duration longer, and many complex wave shapes with more than 3 or 4 spikes—called polyphasic potentials—may appear. Normally, such polyphasic potentials are seen in less than 25 per cent of those recorded in a muscle sampling. Since there will be a reduced number of motor units available for recruitment, interference pattern on strong contraction will not be complete. It should be noted, however, that it takes considerable damage to a nerve or muscle to reduce the recruitment pattern, and the most sensitive test of minimal damage is to observe fibrillations and positive sharp waves.

When the muscle itself is damaged, electromyographic findings distinctly differ from motor neuron damage. Unless the damage is very extensive, the motor units recruited in the muscle resemble the normal electromyograph pattern. As motor units have lost muscle fibers, the motor unit action potentials observed will be smaller than normal. To produce a certain strength of contraction resembling normal strength for a given act, more motor units need to be recruited. Therefore, during electromyographic testing, even a weak contraction will result in full interference pattern. In comparison with disorders of the motor neuron, motor unit potentials seen in muscle disorders are of lower voltage, shorter duration, and of increased recruitment relative to strength of contraction. In addition, variable involvement of the individual muscle fibers result in polyphasic potentials of individual motor unit potentials. At rest, fibrillation and positive sharp waves may be found, especially in inflammatory types of muscle diseases.

It is to be noted that these findings at rest do not distinguish neuropathic and myopathic disorders. However, a further electromyographic finding that helps to distinguish motor neuron from muscular disorders is the presence of fasciculation potentials. These are involuntary and irregularly firing motor unit potentials that generally occur in a random manner when the muscle is apparently at rest. They are believed to be spontaneous discharges within the axon itself, and are observed most frequently in disorders of the anterior horn cell. They are never recorded in muscular disorders. Thus, their presence immediately leads the electromyographer to suspect a diseased motor neuron. *Table I* summarizes the needle electrode electromyographic findings as described above. Finally, there are other spontaneous discharges occurring at rest that have distinctive electromyographic patterns seen in both neuropathic and myopathic disorders with muscle at rest. These have been termed bizarre, high frequency discharges.

Nerve Conduction Studies

In order to determine motor nerve conduction velocity, the nerve is stimulated proximally and distally using a surface or needle electrode to pick up the response from a muscle supplied by the same nerve. Time delay between stimulation and muscle response is called latency of conduction. The distance between the two stimulation points is measured. Then, con-

TABLE I

NORMAL AND ABNORMAL ELECTROMYOGRAPHIC FINDINGS

	Normal	*Neuropathy*	*Myopathy*
Insertion	Normal	Prolonged (positive waves)	Sometimes prolonged
At rest	No activity	Positive waves Fibrillations Fasciculations Bizarre high frequency discharges	Sometimes positive waves and fibrillations Myotonic discharges Sometimes bizzarre high frequency discharges
Minimal contraction	Biphasic and triphasic waves	Increased amplitude Increased duration Polyphasic potentials	Decreased amplitude Decreased duration Polyphasic potentials
Maximal contraction	Full interference pattern	Reduced interference pattern or no interference pattern	Easily obtained interference pattern

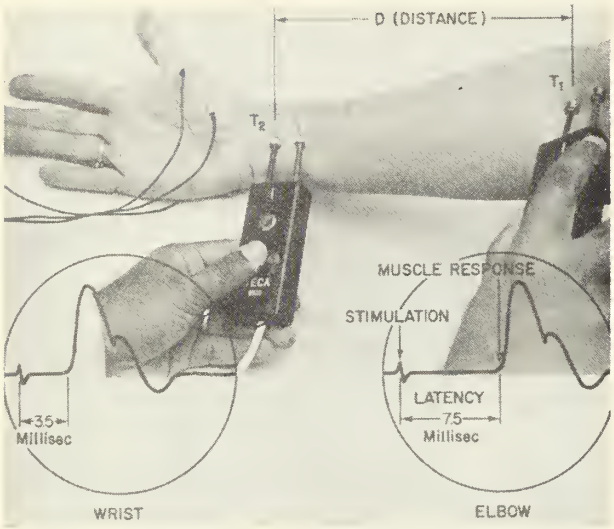


Figure 4. Typical findings in motor nerve conduction studies of median nerve.

duction velocity is calculated according to the formula $CV = \frac{D}{T_2 - T_1}$. CV = conduction velocity in meters/second; D = distance from proximal to distal stimulation in millimeters; T_2 = proximal latency in milliseconds; T_1 = distal latency in milliseconds *(Figure 4)*.

Motor conduction velocities are measured most frequently in superficial nerves, such as median and ulnar nerves in the upper extremities and the posterior tibial and common peroneal nerves in the lower extremities. In general, conduction velocities in upper extremities are faster than in the lower extremities. The average conduction velocities of motor nerves in the upper extremities are 50-60 meters/second. With nerves such as the femoral, where it is difficult to stimulate at two sites, distal latency can be recorded and the responses then compared with normal ranges of values. However, distal latency recordings have not been generally useful in diagnosis as motor conduction velocity.

Sensory conduction latencies and velocities can also be determined by recording sensory impulses with electrodes placed directly over the sensory nerve rather than over the muscles. This is technically more difficult, as the responses are only 20-30 microvolts in amplitude (compared with 5-15 millivolt recordings from the muscles). Sensory nerves routinely checked are median, ulnar, and sural nerves. Pathological slowing of conduction velocities occurs in all types of peripheral neuropathies involving the axonal portion of the neuron. The conduction velocity across the compressed nerve is increased in entrapment syndromes, thus giving a valuable guide to where surgical decompression may be effective. The amplitude and shape of the nerve and muscle responses are also changed in various pathological conditions providing further objective clues to the nature of the underlying pathological process.

Clinical Application

Some common conditions where EMG is particularly useful and should be referred to the electromyographer are as follows:

Anterior Horn Cell Disease. This includes poliomyelitis, amytrophic lateral sclerosis, Werdnig-Hoffman disease, and some myelopathies secondary to cord compression. Fibrillation potentials appear 18-21 days following the death of anterior horn cells. The number of action potentials elicited from affected muscles decreases, and after one-two months action potentials are increased in amplitude and duration. Fasciculation potentials are very often seen in anterior horn cell diseases even when fibrillations and positive waves are not evident. The motor conduction velocity is normal or low normal in anterior horn cell disease, becoming slow only in very advanced disease states.

Axonal Disease. Typical disease conditions primarily affecting axons include Guillain-Barré syndrome, diabetic neuropathy, uremic neuropathy, Charcot-Marie-Tooth disease, and various toxic neuropathies. Axonal disease is differentiated from anterior horn cell disease by the reduced motor conduction velocity. The electromyogram exhibits a reduced number of motor unit action potentials and an increased proportion of polyphasic potentials. Increased amplitude and duration of potentials may be seen, but not as frequently as in anterior horn cell disorders. Fibrillation and positive sharp waves may be seen to varying degrees but are often absent.

Entrapment or Compression Syndromes. These include the carpal tunnel syndrome and ulnar tunnel syndrome at the wrist, ulnar nerve entrapment at the elbow, compression of peroneal nerve behind the head of the fibula, and tarsal tunnel syndrome at the ankle. Nerve responses in both motor and sensory electromyographic readings across an area of entrapment show marked slowing. For example, in carpal tunnel the normal motor latency between stimulation of the median nerve at the wrist and pickup of the response from the thenar muscles is less than 5 milliseconds. In typical carpal tunnel syndrome, the response will be of considerably lower voltage than normal and the latency may be as high as 8-10 milliseconds. By using needle electromyography and both sensory and motor latency studies, the condition can be diagnosed with accuracy in 95 per cent of the cases.[1]

Myoneural Junction Disorders. Myasthenia gravis is the most common example of myoneural junction disease. On repetitive stimulation of the nerve, the muscle

action potential will show a gradual reduction in the amplitude, but nerve conduction velocities will be normal. By using provocative tests with drugs or exercises, the nature of the neuromuscular block can be well defined by electromyography. In fact, such conditions cannot be actually assessed without the aid of the electromyographic examination.

Muscle Diseases. Diseases primarily affecting the muscles produce needle electromyographic findings as described earlier. Examples are muscular dystrophy, myotonia, polymyositis, metabolic myopathies, and steroid induced myopathies. In milder forms of these diseases, the only findings may be an increased proportion of polyphasic motor unit potentials, so the electromyogram is not always helpful in the diagnosis. In myotonic states with an unstable membrane of the muscle and when the muscles fail to relax effectively, a characteristic sound occurs on needle insertion which is described as that of a diving airplane. Therefore, this electromyographic finding is particularly helpful in diagnosis of myotonias. In contrast to most neuropathic ailments, sensory and motor velocities are always normal in these disorders.

Peripheral Nerve Injuries. The distal segment of injured nerve will undergo Wallerian degeneration. Once this occurs, the muscle supplied by the nerve will exhibit fibrillation and positive sharp waves. With complete lesions, motor unit potentials cannot be elicited (either voluntarily or on nerve stimulation). In old lesions or after regeneration, motor unit potentials will be of high amplitude and prolonged duration. During reinnervation, a characteristic polyphasic potential known as nascent potential appears on volition.

Root Compression. Any compromise of the motor root may result in electromyographic abnormalities in the muscle supplied by the root. Included are muscles innervated by the anterior primary ramus (in extremity) and the posterior primary ramus (the paraspinal muscle). The findings include fibrillation and positive sharp waves, and increased number of polyphasic potentials with incomplete interference pattern. Nerve conduction velocity in peripheral nerves will be normal, although amplitude of responses may be reduced in affected muscles.

Plexus Injuries. By examining the samples of muscles supplied by various nerves emerging from the plexus, localization of the injury can be made. For example, in a brachial plexus injury, it is possible to determine whether the lesion is proximal to the plexus, in the trunk, cord, or nerves distal to the plexus.

Discussion

The above brief description covers most of the main points concerning clinical electromyography. The most useful information for the referring physician is in the specific summarization of the nerve conduction and needle electromyographic findings.

Many patients worry about the pain and discomfort associated with EMG. A typical test takes approximately one hour, and includes stimulation of two or more nerves and needle electrode examination of 10-20 muscles. Although the test is uncomfortable, it is by no means intolerable, and most patients have felt that it is less discomforting than a myelographic examination.

A study of electromyography should not be undertaken in a crash course of one-two weeks. It takes a physician at least several years of constant training and practice to become a competent electromyographer. EMG should be performed only by a licensed physician with an academic background and training in medicine: the background training of other personnel—such as physical therapists—is inadequate to perform this test accurately. This is particularly true in today's climate of malpractice, as an inadequate or inaccurate report may lead to serious consequences such as failure to locate a pathological lesion that requires surgery. It should also be remembered that this is only a sampling technique or an electronic extension of the neurologic examination and is not independently diagnostic.

The American Association of Electromyography and Electrodiagnosis has established standards of performance of EMG. In ordering the test, to help ensure quality, a physician should determine whether the electromyographer is a member of this organization. Furthermore, newer techniques, such as recording reflex responses and recording for single muscle fibers, are constantly under development. Only those physicians with special interest in electromyography can expect to perform the new tests competently and completely.

Summary

Clinical electromyography is a useful extension of the neurologic examination for diagnosing and prognosing disorders of the neuromuscular system. Most cases referred for electromyography have possible nerve root compression syndromes, peripheral nerve disorders, or various disorders of the motor unit. The test, which takes about an hour, is only moderately painful and generally includes both nerve conduction studies and needle electrode examination. The test must be done by a qualified physician to ensure accurate results. Serious problems have arisen from electromyography performed by unqualified persons.

(Continued on page 336)

Yeast Vaginitis

Nystatin vs Monistat in Treatment of Vaginal Candidiasis

WILLIAM J. CAMERON, M.D., *Kansas City, Kansas*

A CLINICAL STUDY was conducted at the University of Kansas between January 1, 1976, and May 12, 1977, to determine the safety and efficacy of Nystatin Vaginal Suppositories* in the treatment of monilial vulvovaginitis. The results were compared to a parallel group using Monistat Vaginal Cream** (miconazole nitrate 2%) for the treatment of monilial infection.

Fifty-four patients with initial vaginal cultures positive for candida completed all evaluations. The patients were 16-42 years of age with a mean age of 29.3 years. At the initial visit, a diagnosis of candidial vaginitis was confirmed by the presence of clinical signs (erythema, swelling, excoriation, ulceration, discharge), the presence of pseudohyphae on a KOH smear taken from vaginal scrapings, and a positive culture for candida (Nickerson's media). Patients were informed as to the nature of the study and written consent was obtained prior to entry into the study. The medication was dispensed in accordance with a pre-determined randomization chart, and patients were instructed to insert the suppository or cream into the vagina each morning and night for 14 days. Patients receiving the cream were instructed to wear a pad during the treatment period to reduce rapid discharge of the medication. Each patient was requested to maintain a chart of symptoms (itching, irritation, burning, discharge, odor, dyspareunia) and evaluate their symptoms each day as either none, mild, moderate, or severe. These were usually recorded for the 14 days of treatment and seven days post-treatment.

Gynecologic examinations were performed initially, 14 days after inception of therapy, one week after treatment, and one month after completion of treatment. The therapy was evaluated on the final visit as either a cure (asymptomatic with negative post-treatment cultures), a failure (persistence of signs or symptoms of vaginitis, or positive post-treatment cultures), or symptomatic relief (completely asymptomatic with one or more positive cultures, post-treatment).

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** Ortho Pharmaceutical Corporation, Raritan, New Jersey.

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Nystatin vaginal suppositories were compared to Monistat vaginal cream for safety and efficacy in two groups of patients in a 14-day regimen for monilial vulvovaginitis. Analysis of patients' daily records is presented, revealing similarity as to rate of relief and equivalence as to success.

Twenty-seven patients receiving the cream had an initial examination verifying monilial vulvovaginitis by the presence of associated signs and a positive culture. The 26 who completed the series had an age range of 16-40 years with a mean age of 29.1 years. The major complaint noted at the time of the initial examination was itching. At the initial clinical evaluation, essentially all of the patients exhibited erythema and discharge, and approximately one-half had swelling associated with their monilial vulvovaginitis *(Figure 1)*. Of the patients completing the series with this medication, eight were using birth control pills, one was using a diaphragm as a contraceptive, and three patients were pregnant upon entering the study.

Thirty-three patients receiving the suppository had an initial examination verifying monilial vulvovaginitis by the presence of associated signs and a positive culture. The 28 who completed the series had an age range of 22-42 years with a mean age of 29.4 years. As in the alternate therapy, the major patient complaint on initial examination was itching. The clinical evaluation of the initial signs noted in this group was essentially identical to that in the group receiving the cream therapy *(Figure 1)*. Of the patients completing the series using the suppositories, nine were using birth control pills, one was using a foam as a contraceptive, one was surgically sterile, and four were pregnant upon entering the study.

The degree of itching, irritation and burning, as recorded by the patients in daily charts, can be seen in *Figure 2*. Only patients reporting these symptoms and those clinically cured at the end of the evaluation were considered for the study. For each symptom graphed,

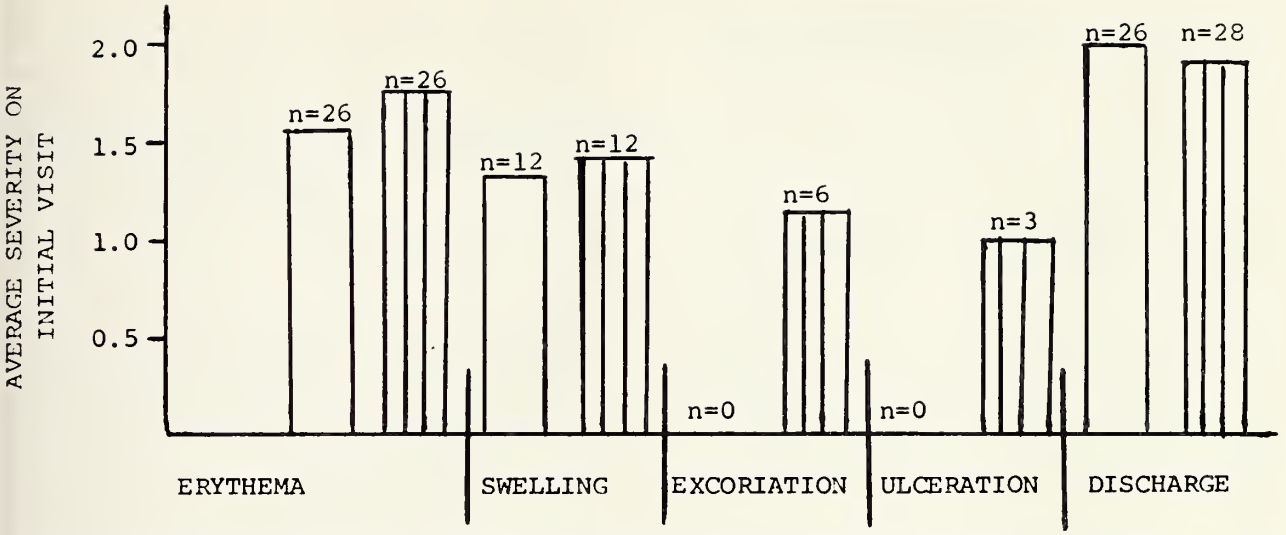


Figure 1. Comparison of signs on initial visit prior to therapy. Note: Each sign category represents the mean severity for those patients (n) with a rating of ≥ 1 on the initial visit and who completed the study series. Ratings included 1 = mild, 2 = moderate, and 3 = severe.

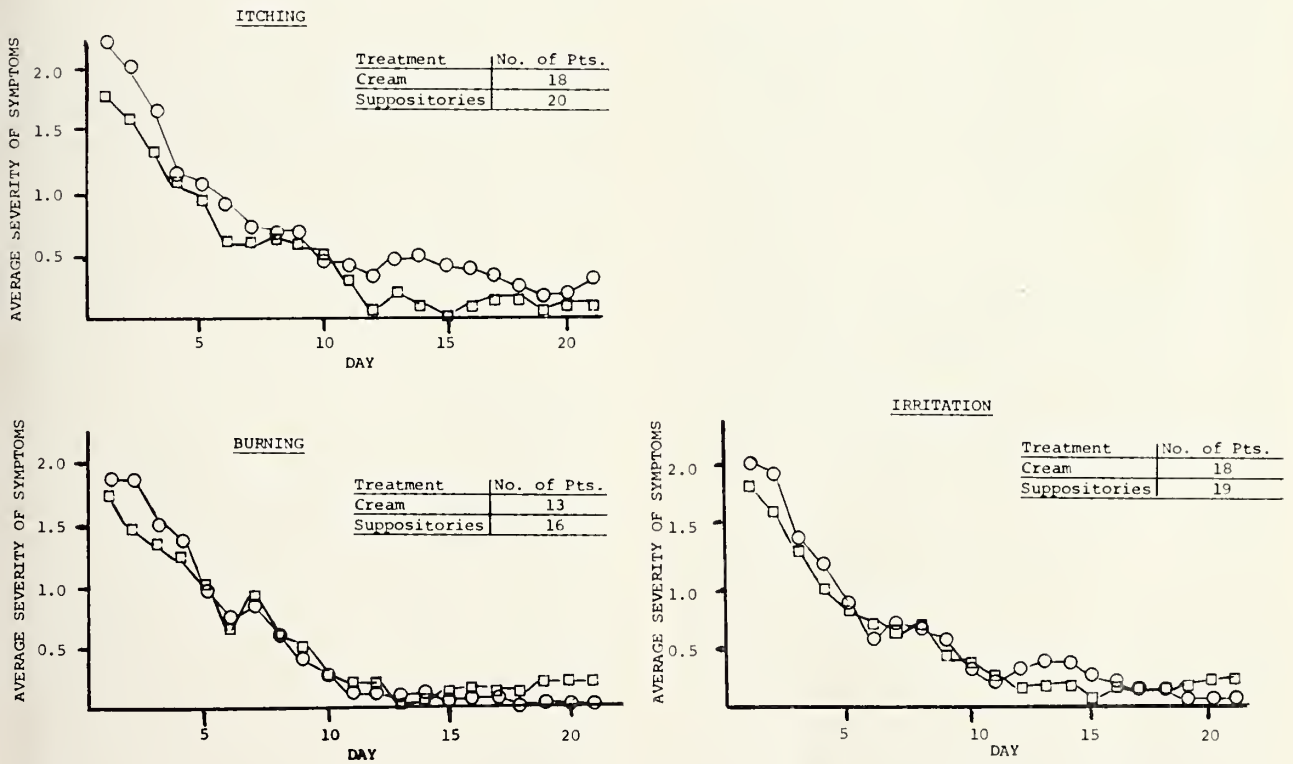


Figure 2. Relief of symptoms recorded by the patients. Note: These graphs were compiled from patients who noted a symptom(s), irrespective of severity, for at least 2 consecutive days during the first four days of treatment. Severity ratings of 0, 1, 2, and 3 represent a patient response of none, mild, moderate and severe, respectively. (□—□ Cream, ○—○ Suppository)

(Continued on page 344)

Carotid Endarterectomy

Surgical Intervention for the Prevention and Alleviation of Strokes

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STROKES were generally thought to be caused by thrombosis or rupture of an intracranial vessel, and that little could be done to prevent or actively treat these catastrophes. As early as 1951, a few bold surgeons, who recognized that some strokes were associated with occlusion of extracranial vessels—and these are accessible in the neck—attempted surgical approaches to the problem.[1] In the mid-1960s, studies reported that 75 per cent of patients admitted to hospitals with signs of cerebral ischemia had surgically accessible extracranial lesions.[2] In one stroke registry, 27 per cent of patients with thrombotic stroke died within 30 days of onset; many others had disabling residual effects.[3, 4]

During the period when the general concept of the causes and evolution of stroke was changing, the surgical attention was being directed to preventive surgical procedures because it was recognized that operations for completed strokes are "too little, too late," and may make the patient's condition worse rather than better.[4]

To understand how prevention can be accomplished, the pathology in these patients must be considered. Those who suffer strokes usually have generalized arteriosclerosis and are often hypertensive. Their coronary artery, and other disease, may be as much a limitation to survival as the cerebral problem. Many have aortic and peripheral aneurysms and occlusions. These factors must always be taken into account in planning treatment. More than half have multiple involvement of the cerebral vessels.[5, 6]

From the standpoint of clinical symptoms and physical findings, how are patients diagnosed at a stage when they can be helped by surgery? From 30-75 per cent of patients are reported to have one or more transient ischemic attacks (TIA) over a varying period of time before experiencing a frank stroke. TIA is a manifestation of local decrease in blood flow to a region of the brain translated into symptoms reflecting loss of function of that particular area. Among the most common symptoms are:

1. Transient blindness (amaurosis fugax), hemianopsia, or scotomata on the side of the vascular lesion;
2. Weakness, numbness, or loss of function of one or both contralateral extremities;
3. Slurred speech or aphasia;

Emerging concepts of cerebral vascular disease, that facilitate alleviative and preventive surgical procedures, are explored; indicative guidelines and surgical techniques are discussed.

4. Dizziness and ataxia.

In considering transient ischemic attacks, one might ask how transient is "transient?" Various authors differ, some defining a few minutes as being transient, and some considering 24 hours of residual neurological deficit as transient.[7]

The next consideration is that of pathology, which results in partial or temporary stroke which improves over a period of days or weeks, reaches a plateau leaving residual deficits, or continues to progress. At the far end of the spectrum, there is the frank completed stroke of sudden onset which may be fatal or result in serious residual effects.

The way in which a stroke evolves is dependent upon the anatomy of the regional circulation and the individual pathology and distribution of disease which is usually related to an arteriosclerotic occlusion or an embolic process, or both, in the non-hemorrhagic group.

The blood supply to the brain evolves from two internal carotid arteries located anteriorly in the neck, and supplying about 90 per cent of the normal circulation; and two vertebral vessels located posteriorly in the neck in a rather inaccessible location for surgical intervention, but supplying only about 10 per cent of the normal demands. Fortunately, many of the lesions most responsible for symptoms occur in or near the readily accessible carotid bifurcation.[6]

Within the cranium, in the circle of Willis, patients with completely normal anatomy have remarkable col-

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lateral pathways not only from anterior to posterior but also from side to side. Some few individuals congenitally lack some collaterals to complete the circle of Willis, or collaterals may become occluded by an arteriosclerotic or embolic process. Other collateral pathways include leptomeningeal vessels and branches of the external carotid communicating with branches of the internal carotid by way of the orbit. There are also communications between the subclavian branches, anterior spinal arteries, occipital branches of the external carotid with anastomosis to the vertebral arteries, and the thyrocervical trunk through the inferior and superior thyroid and ascending cervical vessels. The collateral supply via the external carotid may be so important that, in selected patients with occlusions of several other vessels, endarterectomy of the external carotid artery on one or both sides may be indicated.[4] Collateral blood supply is frequently the determining factor in whether a patient will have a frank stroke, or symptomatic disease which can be treated successfully after timely diagnosis.

It has been said that vertebro-basilar insufficiency, which produces symptoms of weakness and paresthesia, is more likely to involve both sides and produce visual defects which may be bilateral with occasional diplopia, vertigo, and nystagmus.[6] However, when one is considering a patient for surgical treatment, one must not be discouraged from operating upon stenotic lesions of the internal carotid system because symptoms are said to be on the basis of vertebro-basilar decrease in flow due to the rich collaterals around the circle of Willis. As long as collaterals are present, symptoms may be alleviated by opening any vessel which will increase the blood supply to the brain, sometimes on the contralateral side, if that is the only operable vessel demonstrated.

In patients who do not present with symptoms of cerebral vascular insufficiency, carotid stenosis may sometimes be confirmed during routine physical examination through stethoscopic detection of a bruit, a condition which occurs in approximately 75 per cent of stenotic lesions of the internal carotid artery.[6] Another fortuitous finding is reduction of pressure in one or both eyes due to carotid and ophthalmic artery occlusions which may be detected by routine eye examination.

When patients present significant symptoms associated with signs of carotid artery disease, the ultimate evaluation is dependent upon arteriography demonstrating the four vessels supplying the brain and the circulation in the cranium. While there is some risk associated with angiography, in carefully selected patients who demonstrate the proper symptoms and findings, the risk is minimal compared with the risk of cerebral infarction for the untreated patient.

Other non-invasive but less definitive techniques consist of radionuclide flow studies, Doppler directional flow studies of supraorbital blood, and other Doppler studies predicting cerebral flow.

In one controlled series,[4] 102 patients with asymptomatic bruits not operated had an incidence of 19 per cent frank strokes while being studied over a period of several years; while in 119 patients asymptomatic after elective operations there was a 1.7 per cent neurological deficit secondary to operation, and only 1.7 per cent long term non-fatal stroke, and no fatal stroke. There was no surgical mortality.

Patients commonly accepted for surgery include:[1, 4, 6, 8]

1. Those exhibiting TIA in the presence of demonstrable lesions on arteriography;
2. Selected patients with stable strokes;
3. Asymptomatic patients with bruit in the neck and angiographic findings of advanced disease;
4. Those suffering chronic cerebral ischemia associated with demonstrable lesions on angiography.

Commonly accepted contraindications to surgery include:[1, 4, 6, 9]

1. Acute stroke. It is preferable to devote two-six weeks to angiographic and other studies before consideration of surgical treatment;
2. Progressing or unstable stroke;
3. Severe intracranial disease;
4. Severe systemic disease, such as advanced coronary artery disease and advanced kidney disease.

Results

Symptomatic improvements with few postoperative TIAs are reported in 80-90 per cent of patients. The surgical mortality in experienced hands is two per cent or less. The incidence of stroke is markedly reduced when compared with a control series.[6] Fewer than five per cent of patients to date have demonstrated recurrent stenosis and this is detected on an average of five years postoperatively.[10] Personality and cognition are improved in many patients.[11]

Surgical Technique

Atheromatous plaques are removed from the carotid bifurcation and—most importantly—from the internal carotid artery by incision of the vessel near the plaque and its removal. It ordinarily shells out leaving a clean pseudointimal surface. Several years ago, the use of a patch graft was advocated to widen the carotid bifurcation and the origin of the internal carotid with a piece of dacron or saphenous vein. This procedure has been abandoned following the development of a significant number of false aneurysms requiring reoperation.[12]

There are schools who shunt the arteries during the surgical procedure, by placing a small flexible plastic tube within the vessel beyond and proximal to the area being operated upon, to allow continuous flow of blood to the brain.[4, 8] There are some who never shunt these cases during surgery; and there are some who selectively shunt a few of them.[13] Some determine whether to shunt by the arteriographic findings and the number and extent of lesions reducing blood flow; others measure back flow from the internal carotid artery during artery occlusion at the time of operation to determine whether there is sufficient collateral blood supply. These measurements vary, some may be inaccurate, and different authors vary as much as 50 per cent on the back pressure which they use as a criterion for the necessity of a shunt.[14, 15]

There is considerable concern on the part of some observers that division of the carotid body may produce a risky postoperative hypertension, especially in elderly patients. The carotid body may or may not be taken down during endarterectomy.[16]

Deep hypothermia has been employed for a period of time by some surgeons to reduce oxygen requirements during surgery, and allowing safer margins of occlusion time of the carotid artery. If the hypothermia is too profound, cardiac arrhythmias and a drop in blood pressure may result. Systemic heparinization during the procedure is rather universal. Heparin may or may not be reversed at the end of the procedure.

For the past five years, this author has employed the following method of carotid endarterectomy at the carotid bifurcation.

After routine exposure of the common, internal and external carotid vessels, the patient is systemically heparinized. The three vessels are occluded with vascular clamps. An incision is made at the origin of the external carotid artery, ordinarily at the level of the superior thyroid takeoff, and extended down the common carotid artery for a distance great enough to expose the bifurcation and the internal carotid orifice. Endarterectomy is initiated in the line of incision or at the internal carotid orifice removing the plaque and relieving the internal carotid obstruction. The extent of endarterectomy in the common and external carotid vessels is determined by the individual pathology. Following endarterectomy and inspection of the intima, backflow is checked and vessels are flushed. The artery is flooded with heparinized saline. A small Satinsky clamp is placed along the line of incision, and the other clamps are removed allowing blood flow to the brain by way of the internal carotid artery while the suture line is leisurely completed external to the Satinsky clamp. By reestablishing blood flow to the brain during closure of the incision, occlusion time is reduced. Any decrease in diameter along the suture line occurs in the external and common carotid arteries, and leaves the internal carotid vessel with a normal or above normal diameter after endarterectomy, obviating the necessity of a patch graft. In a number of patients total occlusion time has been three minutes or less. A shunt is employed only in highly selected patients as it may be traumatic to the intima of the internal carotid artery. Without a shunt we have seldom had an occlusion time of more than 12 minutes, and in the last two years we have employed hypothermia beginning at the time of anesthetic induction to a core temperature of approximately 34C. This level of hypothermia does not result in hypotension or the production of arrhythmias and its employment seldom slows down the procedure. The carotid sinus nerve is injected with one per cent Xylocaine during the operative procedure to prevent reflexes resulting in hypotension. The carotid sinus nerve is usually preserved during this procedure. Late postoperative angiograms have shown normal configuration and caliber of the internal carotid arteries in those patients studied.

Summary

The concept of stroke has changed in recent years, and we more frequently recognize cerebral vascular disease at a stage when a surgical procedure can be employed to alleviate symptoms and neurological deficit, or more importantly, to prevent future strokes. A review of some of the clinical and pathological vicissitudes has been presented. A guideline of indications and contraindications for operation was reviewed and a unique approach to the technical removal of abnormal plaque at the carotid bifurcation has been presented, whereby incision for endarterectomy is made along the external and common carotid vessels resulting in no narrowing of the internal carotid artery, and reducing occlusion time by reestablishing blood flow during closure of the arteriotomy using a small Satinsky clamp.

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Hemorrhagic Complication

Severe Thrombocytopenia With Subarachnoid Hemorrhage in Infectious Mononucleosis

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MILD THROMBOCYTOPENIA frequently occurs during the course of infectious mononucleosis, but severe thrombocytopenia (platelets < 20,000/cu mm) with hemorrhagic complications is distinctly uncommon. The incidence of this complication in mononucleosis is reportedly less than one per cent.[1] Even more rare is the occurrence of intracranial hemorrhage secondary to thrombocytopenia in infectious mononucleosis. Reported here is the case history of a patient with serologically proven infectious mononucleosis, whose clinical course was complicated by the development of severe thrombocytopenia and subarachnoid hemorrhage.

The occurrence of severe thrombocytopenia with hemorrhagic complications is uncommon in infectious mononucleosis. Reported here is a case complicated by the development of severe thrombocytopenia resulting in subarachnoid hemorrhage in which therapy with corticosteroids led to dramatic clinical and hematological improvement.

Case Report

A 20-year-old male was admitted to the Veterans Administration Hospital, Topeka, in June 1976, with complaints of fever and sore throat of one week's duration. Physical examination on admission revealed the patient to be toxic and febrile with temperature, 38.5C; respirations, 20/min; blood pressure, 110/80 mm Hg. Examination of the throat revealed a congested pharynx. No tonsillar enlargement or exudates were noted; no cervical lymphadenopathy or neck rigidity was present. The remainder of the physical examination was unremarkable.

Throat and blood cultures were obtained on admission, and Ampicillin IV, 1 gm/six hrs, was administered. Initial laboratory data revealed the following results: hemoglobin, 15 gm/100 ml; hematocrit, 45%; WBC, 6,900/cm mm, with a differential of bands, 5%; 26% polymorphonuclear; lymphocytes, 62%; monocytes, 6%. Platelet count on admission was 34,000/cu mm, several atypical lymphocytes were observed on the peripheral blood smears. Other tests revealed SGOT, 257 (7-40); LDH, 644 (100-225); alkaline phosphatase, 303 (30-85); total bilirubin serum, 1.5 mg/100 ml. Results of chest x-ray and urinalysis were normal; febrile agglutinins and hepatitis-B antigen, negative; monospot test, ordered in view of pharyngitis and atypical lymphocytes observed in the peripheral blood, was strongly positive. Two days after admission, when blood and throat cultures were reported negative, Ampicillin therapy was terminated.

However, a few days after admission, the patient continued febrile and complained of sudden onset of severe progressive headache. Fever persisted at 38-40C. Platelet count dropped to 11,000/cu mm. He developed a fine petechial rash and became progressively lethargic. Partial thromboplastin time, plasma fibrinogen level, and fibrinogen split products level were all normal. Gingival biopsy did not show any evidence of thrombotic thrombocytopenic purpura. Bone marrow revealed increased cellularity with increased megakaryocytes. There was slight increase in lymphocytes, but the erythroid and granulocytic development was normal. Antiplatelet antibodies could not be demonstrated. Heterophil titer was found to be positive in 1 : 1792 dilution.

The platelet count dropped to 7,000/cu mm the following day, and additional petechial rash developed over the skin and mucosa of the mouth. The patient became semicomatose and a thorough neurological evaluation again failed to disclose a focal neurological deficit. A lumbar puncture was then performed which revealed the cerebral spinal fluid (CSF) to be under increased pressure. CSF was grossly and uniformly bloody in all

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three tubes collected. Tests at this time revealed total protein, 134 mg/100 ml; WBC, 142/cu mm with 2 neutrophils and 140 lymphocytes; RBC, 17,000/cu mm; supernatant fluid was xanthochromic after centrifuge. Other test results included CSF glucose, 73 mg/100 ml; simultaneous blood glucose, 90 mg/100 ml. The results of Gram's stain, India ink preparation, and cultures of CSF were negative. The increase in mononuclear cells and protein content of CSF could be attributable to aseptic meningeal reaction following meningeal epistaxis.

The patient was immediately started on hydrocortisone IV (Solucortef) 100 mg/6 hrs. The patient's level of consciousness improved within 48 hours, and the platelet count increased from a baseline value of 7,000/cu mm to 40,000/cu mm. The patient was then administered oral Prednisone 60 mg/day. Gradual clinical and hematological improvement was noted over the next few days. The patient became completely asymptomatic and the platelet count rose to 70,000/cu mm two weeks after steroid therapy was initiated. He was discharged on a regimen of Prednisone 60 mg/day.

The results of a brain scan and electroencephalogram performed prior to discharge were normal. Viral serology (performed twice during the course of hospitalization by the Viral Oncology Lab, Atlanta, GA) revealed positive antibodies to Epstein-Barr Virus in titers of 1 : 640 (ox cell hemolysis) and 1 : 100 (indirect fluorescent antibody technique).

The patient was followed in outpatient clinic over the next several months. A premature attempt one week after discharge to lower the steroid dose was followed by a prompt fall in the platelet count to 40,000/cu mm. However, upon reinstituting Prednisone 60 mg/day, the platelet count returned to 140,000/cu mm. The steroid dose was then cautiously decreased during the next several weeks, and Prednisone was discontinued three months after discharge. The platelet count continued to remain normal several weeks after the complete cessation of corticosteroid therapy.

Discussion

Although fewer than 40 cases of severe thrombocytopenia in infectious mononucleosis have been reported in the literature thus far,[1, 2] this complication is being increasingly recognized. Perhaps many such cases go unreported, particularly when the thrombocytopenia is not accompanied by clinical bleeding. Occurrence of severe thrombocytopenia simulating acute idiopathic thrombocytopenic purpura (ITP) has been reported in other viral illnesses such as mumps varicella, infectious hepatitis, and cytomegalovirus disease.[3-6] Such complications have also resulted from vaccination with live virus.[7, 8]

The mechanism of thrombocytopenia in infectious mononucleosis appears to be immune-mediated, leading to peripheral destruction of platelets. Occurrence of increased megakaryocytes in the marrow in the face of severe thrombocytopenia in the peripheral blood, and the response to corticosteroid therapy as observed in typical cases of ITP, favor this mechanism. Anti-platelet antibodies have been demonstrated in a few instances.[9, 10]

Most of the cases of severe thrombocytopenia occurring in infectious mononucleosis have responded to steroid therapy. However, a few cases which were refractory to corticosteroids have responded to splenectomy.[2] Thus, the overall prognosis of this complication in infectious mononucleosis remains good, although an occasional patient can succumb to cerebral hemorrhage.[11] The occurrence of severe thrombocytopenia (platelet count $<20{,}000$/cu mm) in infectious mononucleosis should be considered as strong indication for commencing corticosteroid therapy, since the incidence of spontaneous hemorrhage below this platelet count is greatly increased.

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Current COMMENT

Acute Bacterial Meningitis

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MENINGITIS denotes an inflammatory reaction involving the meninges without inplication of cause. Prior to the late 1930s, the mortality from bacterial meningitis was greater than 90 per cent, and those who survived often had neurologic defects. Since then, therapy with antimicrobial agents has markedly reversed the previously grim prognosis. For therapy to be effective, meningitis must be suspected early, diagnosed accurately, and treated without delay.

A review of six reported series, spanning a quarter-century (1949-1973) and involving 1,876 cases of purulent meningitis, revealed *Streptococcus pneumoniae, Neisseria meningitidis,* and *Haemophilus influenzae* to be the most frequently encountered organisms in culture-proven bacterial meningitis. However, in one quarter of the cases, a bacterial cause was suspected, but not confirmed. Although *Streptococcus* spp., *Staphylococcus* spp., and gram-negative bacilli account for less than one-sixth of all cases, recent data suggest that these organisms are becoming a more frequent cause of meningitis, especially in hospitalized patients.

The peak incidence of bacterial meningitis occurs during the neonatal period. However, the peak incidence for *H. influenzae, St. pneumoniae,* and *N. meningitidis* meningitis occurs from 3-18 months of age.

There is a distinct relationship of age to the causes of bacterial meningitis. In the neonatal period, the enteric organisms, Group B β-hemolytic streptococci, staphylococci, and *Listeria monocytogenes* are the usual causes of bacterial meningitis. *H. influenzae, N. meningitidis,* and *St. pneumoniae* cause the majority of meningitis between the neonatal period and five years of age. From 5-20 years, *St. pneumoniae* and *N. meningitidis* are the most common causes of bacterial meningitis. Above age 20, *St. pneumoniae* is the most common cause of meningitis, and after age 50, the enteric organisms and staphylococci must also be considered.

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Presentation

The clinical presentation of neonates and infants with meningitis is non-specific and the physician must have a high index of suspicion in order to make this diagnosis *(Table I).* Children and adults usually present with one

TABLE I

ACUTE BACTERIAL MENINGITIS: CLINICAL PRESENTATION

A. Neonatal Period	C. Children and Adults:
1. Respiratory distress	1. Fever
2. Fever	2. Vomiting
3. Vomiting	3. Headache
4. Anorexia	4. Altered consciousness
5. Jaundice	5. Stiff neck
6. Irritability	6. Convulsions
7. Drowsiness	
8. Convulsions	
B. Infant:	D. Elderly:
1. Fever	1. "I'm not feeling well," may be the only clue
2. Vomiting	
3. Irritability	
4. Drowsiness	
5. Convulsions	
6. Bulging fontanelle	

or more symptoms or signs that should suggest the possibility of meningitis. In the elderly, the presentation may be just as nonspecific as in the neonate and infant.

The clinical setting of a patient with suspected bacterial meningitis may suggest the cause. Staphylococcal and gram-negative bacillary meningitis are seen sporadically throughout the year, whereas, *H. influenzae, St. pneumoniae, N. meningitidis,* and unknown meningitides have a distinct winter-spring clustering. Males outnumber females except for meningococcal infection in which females may predominate. Staphylococcal and gram-negative meningitides are often hospital-associated, but *H. influenzae, St. pneumoniae, N. meningitidis,* and unknown meningitides are rarely hospital-associated. Antecedent antibiotic therapy for an infection other than meningitis is common in the staphylococcal, gram-negative and unknown groups; less common in the *H. influenzae* and *St. pneumoniae* groups; and unusual in the *N. meningitidis* group. Underlying medical illnesses are common in the gram-negative group, less common in the staphylococcal and *St. pneumoniae* groups; and unusual in the *N. meningitidis* and unknown groups. An identifiable predisposing factor is found commonly in the staphylococcal, gram-negative, *H. influenzae,* and *St. pneumoniae* groups, is found less often in the unknown group, and is unusual in the *N. meningitidis* group.

Diagnosis

Because the clinical presentation is non-specific and often does not suggest the causative diagnosis of meningitis, a lumbar puncture with examination of the cerebrospinal fluid (CSF) is indicated in any patient suspected of having meningitis. There are three CSF profiles associated with meningitis: (1) purulent profile with elevated protein and leukocyte count, predominantly polymorphonuclear leukocytes, and a low glucose; (2) lymphocytic low-glucose profile; and (3) lymphocytic normal-glucose profile *(Table II)*.[1] Other diseases with abnormal CSF findings include brain abscess, malignant involvement of the meninges, meningovascular syphilis, multiple sclerosis, cerebrovascular accident (CVA), and subarachnoid hemorrhage *(Table III)*.

A diagnosis of bacterial meningitis can be further confirmed by examining a gram stain of the CSF. Smears are positive in 55-90 per cent of patients with an incidence of erroneous interpretation of 2-10 per cent. CSF cultures are positive 52-92 per cent of the time, and blood cultures are found to be positive 30-60 per cent of the time. Antibiotic pretreatment probably does not alter CSF findings in bacterial meningitis, but does reduce the chance for diagnosis by CSF gram stain, CSF culture, and blood culture.

TABLE II

CSF PROFILES IN MENINGITIS[1]

A. Purulent Profile
  1. Infectious Causes
    a. Common
      (1) Bacterial Meningitis
    b. Uncommon
      (1) Viral Meningitis*
      (2) Embolic Cerebral Infarction*
      (3) Parameningeal Infections*
      (4) Amebic Meningoencephalitis
      (5) Tuberculous Meningitis
      (6) Acute Hemorrhagic Encephalitis*
  2. Noninfectious Causes (rare)
    a. Chemical Meningitis
    b. Hypersensitivity Meningitis*
B. Lymphocytic Low-glucose Profile
  1. Common
    a. Tuberculous Meningitis
    b. Fungal Meningitis *(Cryptococcus, Coccidioides)*
  2. Uncommon
    a. Bacterial Meningitis
    b. Viral Meningitis
    c. Carcinomatous Meningitis
    d. Sarcoidosis of Meninges
C. Lymphocytic Normal-glucose Profile
  1. Common
    a. Viral Meningitis
    b. Post Infectious Encephalomyelitis
  2. Uncommon
    a. Post Vaccination Encephalomyelitis
    b. Bacterial Meningitis
    c. Parameningeal Inflammation
    d. Fungal and Tuberculous Meningitis
    e. Parasitic Disease

* CSF glucose level normal.

In the last several years, counterimmunoelectrophoresis of serum and CSF has been used to aid in the causative diagnosis of bacterial meningitis.[2, 3] By using antisera directed toward *H. influenzae* type b, *N. meningitidis* groups A, B, C, and Y, and pneumococci (omni-serum), it is possible to demonstrate the presence of specific antigen in serum or CSF in two-thirds of patients who later are culture-confirmed to have bacterial meningitis caused by one of these organisms. The advantage of this test is that the result is available in one-half hour; however, its usefulness is limited to those types of meningitis listed.

More recently, the limulus lysate test has been used to demonstrate endotoxin in CSF, which may be useful in diagnosing gram-negative bacillary meningitis.[4] Other tests have been used to distinguish between bacterial and

TABLE III
CSF FINDINGS IN VARIOUS CNS DISORDERS

CSF Findings	*Bacterial Meningitis*	*Aseptic Meningitis*	*Brain Abscess*	*Tuberculous Meningitis*	*Cryptococcal Meningitis*	*Malignant Involvement of Meninges*	*Meningo-vascular Syphilis*	*Multiple Sclerosis*	*CVA*	*Subarachnoid Hemorrhage*
Appearance	Cloudy	Clear	Clear	Clear with pellicle	Clear	Clear	Turbid	Clear	Bloody	Xanthochromic (bloody)
Cells										
Type	PMN's*	Lymph's (PMN's early)	Lymph's or PMN's	Lymph's	Lymph's	Lymph's* Malignant	PMN's or Lymph's	Lymph's	RBC's	RBC's
Number/ mm^3	500-1,000	6-1,000	5-50,000	25-500	10-500	10-500	100-2,000	5-50	Variable	Variable
Protein (mg %)	50-1,000	45-100	45-200	45-500	45-100	60-500	50-150	15-80	20-300	> 2,000
Sugar	< 40% of SBS	Normal	Normal	< 40% of SBS	< 40% of SBS	< 50% of SBS	> 50% of SBS	> 50% of SBS	> 50% of SBS	> 50% of SBS

* PMN's = polymorphonuclear leukocytes, Lymph's = lymphocytes, SBS = simultaneous blood sugar, and RBC's = red blood cells.

non-bacterial causes of CSF abnormalities, but none help in the specific diagnosis of bacterial meningitis.

Therapy

When the organism is unknown, antibiotic selection is determined by the patient's age and the organisms that most frequently cause bacterial meningitis in that age group *(Table IV)*. Note that oxacillin should be used when staphylococcal meningitis is suspected; oxacillin and gentamicin should be used when the meningitis is hospital-associated; and ampicillin can be substituted for chloramphenicol if *H. influenzae* sensitive to ampicillin is confirmed as the causative agent.

The treatment of meningitis when the causative agent is known is also listed in *Table IV*. Note that when

TABLE IV
ACUTE BACTERIAL MENINGITIS: THERAPY

A. Organism Unknown*
   1. Neonate
      a. Ampicillin 300-400 mg/kg/IV/24 h
      b. Gentamicin 1.5 to 2 mg/kg/IV/8 h (12 h FIRST WEEK OF LIFE)
   2. Infant
      a. Chloramphenicol† 50-75 mg/kg/IV/24 h
   3. Child
      a. Chloramphenicol† 75 mg/kg/IV/24 h
   4. Adult
      a. Penicillin-G 240,000 units/kg/IV/24 h
B. Organism Known
   1. *St. pneumoniae*
      a. Penicillin-G 240,000 units/kg/IV/24 h
   2. *Streptococcus* spp.
      a. Group A or B
         (1) Penicillin-G 240,000 units/kg/IV/24 h
      b. Group D (Enterococcus)
         (1) Penicillin-G 240,000 units/kg/IV/24 h and Gentamicin‡ 1.5-2 mg/kg/IV/8 h
   3. *S. aureus*
      a. Oxacillin 200 mg/kg/IV/24 h
   4. *Listeria monocytogenes*
      a. Penicillin-G 240,000 units/kg/IV/24 h
   5. *H. influenzae*
      a. Chloramphenicol† 75 mg/kg/IV/ 24 h
   6. *N. meningitidis*
      a. Penicillin-G 240,000 units/kg/IV/24 h
   7. *E. coli, Klebsiella* spp., *Proteus* spp., etc.
      a. Chloramphenicol 75 mg/kg/IV/24 h
   8. *Pseudomonas aeruginosa*
      a. Gentamicin‡ 1.5-2 mg/kg/IV/8 h (Tobramycin‡ 1.5-2 mg/kg/IV/8 h)

* If staphylococcus suspected, use oxacillin, 200 mg/kg/IV/24 h. If hospital-associated, use gentamicin and oxacillin.

† If infection caused by ampicillin sensitive *H. influenzae;* ampicillin 300-400 mg/kg/IV/24 h can be used.

‡ 3-5 mg/day intrathecally, also.

gentamicin or tobramycin are used, concurrent intrathecal therapy is necessary.[5, 6] Ampicillin may be substituted for chloramphenicol in the treatment of *H. influenzae* meningitis if the organism is sensitive to ampicillin. The mainstay of therapy for meningitis in the adult is penicillin-G for pneumococcal, Group A or B streptococcal, listerial, and meningococcal meningitis. In penicillin-allergic patients, chloramphenicol is an adequate alternative. Because the cephalosporin antibiotics do not cross the blood-brain barrier, they should not be used to treat meningitis, and patients treated with a cephalosporin for bacteremia should be observed closely for the development of meningitis.

Therapy for most cases of meningitis should be continued until the patient has been afebrile for five to seven days. Because of the frequent occurrence of relapse with gram-negative bacillary meningitis, therapy should be continued for at least three weeks. CSF cultures obtained on the third day of therapy should be sterile; if not, therapy should be adjusted accordingly. At the end of therapy, there should be less than 40 WBCs present; all lymphocytes, normal sugar, and a normal or near normal protein in the CSF.

From time to time, glucocorticoids have been recommended as an adjunct in the therapy of meningitis. In children, steroids are neither helpful nor harmful, but in adults, their use is associated with an increased mortality. Thus, steroids should not be used in the therapy of meningitis unless adrenal insufficiency is present. Heparin is indicated in patients with fulminant disseminated intravascular coagulation that is not reversible with proper fluid replacement and treatment of the infection.

The aminoglycosides given intrathecally in the lumbar region may not attain therapeutic levels in the cisternal or ventricular fluid. Thus, patients with gram-negative bacillary meningitis who do not respond to parenteral and intrathecal aminoglycoside therapy, or those who relapse after a three-week course of therapy, may benefit from implantation of a ventricular reservoir and therapy via the reservoir.

Prognosis

The prognosis from bacterial meningitis is related to the causative agent, age (young and old are at highest risk), degree of cerebral dysfunction on admission, absence of nuchal rigidity, and the presence of an underlying illness or predisposing factor.[7] Prognosis is also worse in patients with a positive CSF gram stain, CSF culture, or blood culture. In contrast, sex, race, duration of illness prior to diagnosis, and antibiotic pretreatment are not related to outcome.

Prevention

Presently, prevention of meningitis is restricted to that form caused by the meningococcus. Rifampin (5 mg/kg in children < 1 year of age, 10 mg/kg in older children, or 600 mg in adults every 12 hours for 4 doses) is the drug of choice to prevent secondary cases among intimate contacts of patients with meningococcal meningitis.[8] Intimate contacts are those persons who regularly eat and sleep with the patient. Vaccines are available for the prevention of type A and C meningococcal disease.[9] Use should be restricted to epidemic or high-risk situations.

Self-Assessment Questions
(More than one answer may be correct.)

1. The risk of bacterial meningitis is greatest:
   a. in elderly debilitated patients;
   b. in the immunosuppressed host;
   c. among newborn infants;
   d. among military recruits.
2. The signs and symptoms of bacterial meningitis:
   a. are fever, headache, and vomiting with signs of meningeal irritation as the usual presenting complaints;
   b. include Kernig or Brudzinski signs, and their absence virtually excludes the diagnosis;
   c. among infants in the first year of life usually relate to inflamed meninges;
   d. can include suppurative arthritis which may lead to a bacteriologic diagnosis.
3. Match the clinical situation with the antibiotic of choice:
   a. infants less than two months old without a bacteriologic diagnosis;
   b. infants older than two months without a bacteriologic diagnosis;
   c. ampicillin-resistant *H. influenzae;*
   d. gram-negative organisms;
   e. *S. aureus* meningitis;
   f. meningococcal meningitis;
   g. pneumococcal meningitis:
      1. intrathecal aminoglycoside;
      2. chloramphenicol;
      3. penicillin-G;
      4. ampicillin;
      5. systemic aminoglycoside;
      6. cephalothin;
      7. oxacillin or nafcillin.
4. A 20-year-old black man was admitted with a history of a headache of four days and vomiting for two days. He also complained of photophobia, a single

rigor, and fever. He denied mental changes, sore throat, cough, or URI symptoms. On physical examination, BP was 130/80 mm Hg; pulse, 56/min; respirations, 16/min, and temperature 100F. He could not put his chin on his chest and could not put his head between his knees while sitting. Remainder of examination was normal. An LP was done:

CSF protein: 65 mg %
CSF sugar: 67 mg %
CSF leukocyte count: 114/mm³ with 3% PMNs and 97% lymphocytes
Serum sugar: 125 mg %

a. What is the most likely diagnosis?
   1. bacterial meningitis;
   2. viral meningitis;
   3. tuberculous meningitis;
   4. brain abscess.

b. What would you do?
   1. treat symptomatically and place at bed rest;
   2. repeat LP in eight hours;
   3. start penicillin-G immediately;
   4. isolate patient.

5. A 34-year-old man was admitted with a two-day history of headache, stiff neck, lethargy, and difficulty remembering recent events. He had been well until his present illness and had never been hospitalized in the past. On physical examination, BP was 125/80 mm Hg; pulse, 120/min; respirations, 20/min; and temperature, 103F. Patient had positive nuchal rigidity, Kernig, and Brudzinski signs. Remainder of the physical examination was normal. An LP was done:

CSF protein: 125 mg %
CSF sugar: 15 mg %
CSF leukocyte count: 525/mm³ with 93% PMNs and 7% lymphocytes
Serum sugar: 120 mg%

a. What is the most likely diagnosis?
   1. viral meningitis;
   2. pneumococcal meningitis;
   3. meningococcal meningitis;
   4. tuberculous meningitis.

b. What would you do?
   1. treat symptomatically and repeat LP in eight hours;
   2. start penicillin-G immediately;
   3. start chloramphenicol immediately;
   4. isolate the patient.

(Answers on Pg. 336.)

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Carotid Endarterectomy

(Continued from page 326)

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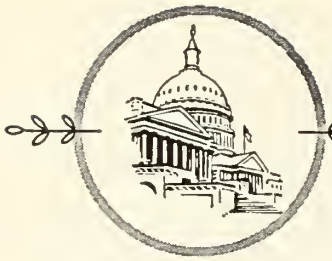
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Socio- ECONOMICS

Considerations Relevant to Incorporation

The decision whether or not to incorporate should be made only after all the advantages and disadvantages have been considered. In many cases the advantages will far outweigh the disadvantages. However, there are still considerations which may preclude or postpone the decision to incorporate.

On the positive side, the economic advantages offered the professional as a "common law employee" of his own corporation are both numerous and significant. These include continuity of business existence, limited liability, liberal qualified pension and profit sharing plans, group term life insurance plans, accident and health insurance plans, wage continuation plans, and a variety of other perquisites the corporation can provide on a tax favorable basis.

Other factors must be considered, however. Professionals face the formal requirements of doing business as a corporation. There must be proper incorporation under Kansas law; meetings must be held and minutes kept, stock certificates issued and allocated, and the effect of the change on patient relations weighed.

More importantly, tax problems must be considered. What should be done with the assets and liabilities of the business upon incorporation? Should *Subchapter S status* be elected? Will corporate profits run afoul of the *accumulated earnings tax* or the *personal holding company penalty tax?* Will dividends have to be declared, thereby incurring the imposition of federal income taxes on both corporate profits and dividend payments? Will the adoption of liberal employee benefit plans result in unreasonable compensation problems?

In order to take advantage of corporate status, decisions will have to be made concerning the types of benefit plans to be provided and the time at which they should be adopted. Should the plans be qualified or nonqualified, pension or profit-sharing, or some combination of these plans? What are the relative costs and tax ramifications, and what design requirements are necessary to insure success?

The effect of changes in the law as accomplished by the *Employee Retirement Income Security Act of 1974*, the *Tax Reduction Act of 1975* and the *Tax Reform Act of 1976* must be considered.

Generally, with proper legal and tax counsel, these problems can be avoided or minimized.

Ultimately, however, the decision whether or not to incorporate will be determined by the tax advantages which will accrue to the professional.

An example of how significant these benefits may be is illustrated below.

For our example we have assumed this professional is earning $75,000 per year after expenses and has elected to incorporate and contribute the maximum permissible to Qualified Benefit Plans.

John Doe, M.D. Not Incorporated			John Doe, M.D.P.A. Incorporated
$75,000	Gross income		$75,000
7,500	Deductible benefits		25,000*
67,500	Net taxable income		50,000
	Individual expenditures		
$22,961	Federal and state income taxes		$13,029†
1,400	Social security taxes		950‡
5,000	Personal insurance plans		Incl. Above
1,000	Miscellaneous		Incl. Above
30,361	Total individual expenditures		13,979
$37,769	Net spendable income		36,021
	Total tax savings	$10,382	
	Increase in Benefit Plans	$17,500	
	Decrease in Net Spendable Income	1,748	
	Gain by Incorporation	$15,752	

* Includes approximately $1,000 operating expenses to corporation

† Assume $10,000 personal itemized deductions

‡ This amount is matched by the corporation

CAUTION: It should be noted that this illustration is of a general nature. Proper legal and tax counsel should be consulted to determine how a professional corporation could benefit you.

How to Find a "Find"

Ed Note: This is the sixth in the series of articles prepared by the AMA Department of Practice Management, Division of Medical Practice. For other articles on this topic, see the following issues: November, 1977, December, 1977, and January, 1978, February, 1978, and March, 1978.

The subject of practice management has become more critical in recent years. To assist the physician in a smoother operation of his office, the Department has produced workshops for medical assistants dealing with such aspects as collections, public relations-telephone techniques and office management, as well as practice management workshops for the new physicians. The Kansas Medical Society Executive Office will present such workshops upon demand by members.

OVER A CUP OF COFFEE after hospital rounds, one of your associates remarks that his new employee recently hired is "a real find." Maybe he was just lucky, but it could be that he used recruiting and hiring techniques that helped him locate an above average applicant for the job.

Higher starting salaries and annual cost of living increases, in addition to deserved merit raises, make it more important than ever for the physician to have top quality staff personnel. With pay scales increasing each year, the doctor cannot afford to hire mediocre help.

The recruiting process should be designed primarily to result in only *qualified* applicants entering your office for the hiring interview. Interviewing any applicant, regardless of how talented, who does not meet your particular requirements, is a waste of time and money.

It is strongly recommended that you prepare a written job description for the position. When you start looking for a new employee there are at least three approaches you can take.

1. Word-of-mouth advertising. Ask colleagues if they are aware of a qualified person for the job you have in mind.

It is wise to limit this to professional circles. If the information is broadcast to the public you may find friends or neighbors applying for the job or referring applicants who are unfitted. Then, when it is necessary to reject them, hard feelings may result.

Your present medical assistant staff may often have information on other medical assistants who are planning to change jobs or who may have been out of the job market for awhile and are now planning to go back to work. Check with your staff, but recognize that if they recommend a person who does not get the position they may be disappointed and could make it uncomfortable for the new person hired. To help avoid this type of situation your staff should know the qualification requirements *(outlined in the job description),* so they will not be tempted to recommend personal friends not able to meet the requirements.

2. If you are in an area where there is a local chapter of the American Association of Medical Assistants (AAMA), the secretary of the association may know of experienced assistants who are looking for employment. Another source you will want to check is the local, two-year junior college. In the past few years, many of these colleges have added associate degree courses in medical assistant training. Eventually, these programs may produce a pool of trained (but not experienced) medical assistants to draw from. At the present time, your best bet is to call the college to find out if potential applicants are available.

3. Advertising is still one of the main vehicles used in recruiting. It is easy and relatively inexpensive to place an ad in your local paper.

Constructing an advertisement demands a critical choice of words. What you say is essential in having the ad serve its intended purpose. If the copy is too general it will bring responses from people who are not qualified for your particular position. The wording of the ad should help screen applicants for you. For example, to advertise for an "assistant" can result in clerical as well as clinical applicants. To advertise for a "secretary" will bring replies from business secretaries as well as medical secretaries. Do you want an experienced person? Or are you willing to train the new employee? If you are going to dictate your medical records on a machine, shorthand is of little advantage—but a medical vocabulary and the ability to spell medical words is a must. If the major typing job is to head-up medical charts and to fill in insurance claim forms, a typing speed of 85 words/min is not important. The 35-word/min typist who can do repetitive jobs accurately is adequately qualified.

The length of the ad and whether it should be in a box or otherwise positioned so that it will attract more attention, at an extra cost, depends largely on the job situation in the area at the time you are looking for help. If it is a tight job market, be prepared to spend more, both for longer copy and better display space. The employment situation will vary considerably from time to time. Sometimes four lines in the middle of a long list of want ads will produce sufficient responses, such as:

HELP WANTED

Medical Office Assistant—Clerical Medical Dictating and Transcribing. Reply by letter only to: Box XYZ Newspaper Anytown, USA.

At another time, with a tight employment market, it

might be appropriate to purchase additional space and write longer, more selling copy:

> Excellent position for person experienced in medical dictation and transcribing. Modern word processing equipment. Requires medical vocabulary. Good working conditions and above average pay scale. Reply by letter summarizing training and experience. Send to Box XYZ Newspaper Anytown, USA.

The ad should require a response that will allow you to eliminate those people whose replies are poorly constructed, perhaps indicating lack of pride in their work. When seeking a person to fill a clerical vacancy you may wish to request a letter rather than a resume. In this way you will have a fair idea of the communication skills of the applicant. When advertising for clinical employees, a resume with a short letter is usually sufficient to help screen replies.

Unless you are in need of a new employee immediately, it is always best to use a box number for reply. To use your name or telephone number will result in interruption of the work schedule to answer requests for interviews or additional information.

After the ad has secured a number of responses, use the telephone to contact the most promising applicants. You will ask about prior employment, availability, and a few other basics previous to arranging an interview, but the phone call is also important to help you evaluate the applicant's voice and manner. Much of the image of the physician and his practice is created by telephone contact between your staff and the public. The people you hire should reflect this professionalism.

When you have screened the applicants down to the top three, the next step is the employment interview. You have completed the recruitment procedure and are now moving into the hiring process—which is a story all of its own. (See *JKMS,* December 1977, p. 516).

Clinical Electromyography

(Continued from page 321)

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Acute Bacterial Meningitis

(Continued from page 333)

Answers to Questions

1. c
2. a & d
3. a. 4 and 5
   b. 2
   c. 2
   d. 1 and 5, or 2
   e. 7
   f. 3
   g. 3
4. a. 2
   b. 1
5. a. 2
   b. 2

WORKMEN'S COMPENSATION PAYROLL AUDITS

Within the next thirty days, field payroll auditors of Casualty Reciprocal Exchange will be calling on those physician offices who have placed their insurance with that organization.

Your cooperation in having the necessary records available for the completion of the audit will be appreciated. The sooner the audits are made, the sooner the Dodson Insurance Group can calculate the earned savings for the past year and make the necessary distribution.

In case you have been asked to provide a voluntary report instead, you can help materially in reaching an early determination of savings by returning the requested information as promptly as possible.

The President's Message

The cost of health care is one of the leading topics of conversation these days. Anyone looking into the subject soon realizes that it is not a simple problem with the single solution that some planners would suggest.

The 1976 Consumer Price Index shows medical care at 184.7 on the CPI (1967 being 100.0). Some say it is the fastest rising item in the CPI. However, a casual look at other services reveals the following rates of increase: repainting the living or dining room, 225.6; repairing washing machine, 200.4; baby-sitting, 214.6; furnace repairs, 207.1. The government's postal charges have increased a healthy 222.3, and if Social Security tax index had been available for 1976, it would have registered in at 308.2, and 332.41 for 1977. Makes us seem like pikers, doesn't it?

Per capita income over the same period, after taxes, was recorded at 202.0.

An impressive array of figures, they prove only what the person utilizing them wants them to prove.

The public seems a little schizophrenic in its attitudes on the cost of health care. Of those responding to a Gallup survey in December 1977, 71 per cent were confident of their ability to pay the cost of health care; 50 per cent felt that they were capable of handling and paying for a major illness. And yet, 61 per cent favored National Health Insurance, knowing that it would increase costs and demands for health services. Our elected officials have found that repeatedly crying "CRISIS" creates that "FACT" in the public's mind. No matter that the government's statistics are not telling the whole story or are biased to get the politicians the power they seek, they are believed by a large number of our patients and fellow Americans. The public must perceive that we care about their concerns over rising medical costs, and the problem this creates for them in their lives.

The AMA Commission on the Cost of Health Care had an impressive membership from many disciplines, and labored hard in its deliberations. Their recommendations in the various areas of the study have pleased no one group or industry, but they do provide a platform or fabric on which to review, analyze, and contemplate the problems and solutions.

While we cannot dictate to the insurance companies that they should have deductibles in their policies to avoid abuses, or offer a variety of programs to groups—nor can we dictate to the hospitals that they should investigate joint purchasing cooperatives, avoid duplication of services, consider ambulatory care centers—we can ask ourselves to review our practice patterns with the idea of cost to our patients. It is imperative that we lead our profession and the public in cost control.

Routine orders may be routinely expensive. I remember Dr. Carleton Matthews remarking during part 2 of the American Board of Surgery exams, "Some people keep making the same mistake for twenty years and calling it experience." Routines usually are an excuse for leaving the brain in neutral.

Are we guilty of admitting patients to the hospital for "checkups"? Some of us are, and by doing so, often for no better reason than our convenience, we are guilty of increasing costs that all of us pay for in higher premiums. The most frequent comment I hear at the medical meetings is that IPPB is the most overused hospital service in the nation! Its benefit to a majority of its recipients is doubted by the experts.

Medical schools and their teaching affiliates are often accused of ordering batteries of tests and procedures in the guise of teaching students, interns, and residents proper care. Recently, requests from various groups and this Society have called for courses regarding costs of tests and education of house staffs on the costs of medical care.

The American Urological Association is holding cost conferences, similar to morbidity and mortality conferences, to review the indications, relevance, and cost of tests done in the diagnosis and treatment of the patient.

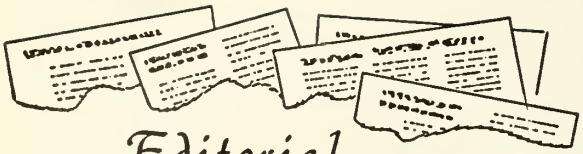
Using our technology in accordance with proper indications would also hold costs in line. Lest I be misunderstood, I am not advocating dictatorial use of CAT scans, sonography, etc., but until new indications and uses for these expensive modalities are proven effective, staying within the established uses seems prudent. Overutilization and improper utilization are wasteful and cannot be justified.

As professionals and physicians, we must demand a higher ethic of ourselves than that which the public imposes on us. We must ask ourselves, "What can I do to hold down costs?" We might be surprised at what we can accomplish without sacrificing quality of care. Don't let our profession be stolen from us by default!

Fraternally yours,

Warren E. Mayer, M.D.

President



Editorial COMMENT

Control Switch

At last report, there was no indication that the spokesmen for the sundry pressure groups were running out of energy in their efforts to expose the deficiencies of the medical profession. This is not a special reference to the President's love-the-sinner-but-hate-the-sin rhetoric of a few weeks ago, when he defoliated the AMA sanctuary but assured us that he didn't mean to singe the individual birds. This sort of hatchet work has always been part of the political scene. We are referring, rather, to those panels, committees, and community meetings which emphasize that the profession has never done much good, is now doing even less and, unless that group's recommendations are adopted, will become an intolerable menace. The popular solution at the moment is to put the patient in control (which translates, in the public application, to letting the pressure groups make the rules).

The seeming reluctance of the physician to accept this committee-promoted control system may stem from the historic base of medical service. It wasn't too long ago that the patient relied for the most part on the physician for opinion and advice. Usually the patient accepted what was offered but any objections or desire to regulate the process was demonstrated by the degree to which he pursued the physician's recommendations. But now, we are informed, the patient is much more knowledgeable about medical matters and is therefore deserving, in the new social order, of the primary role in deciding what should be done on his behalf.

But this "knowledge" which the patient has acquired is chiefly an awareness of methods and techniques, and a superficial acquaintance with a few therapeutic modalities which have led the public not so much to a capability to choose objectively the optimum course for its own care as to an expectation of infallibility from its medical system. The patient's exposure to lay presentations of medical matters and syndicated medical interpretation has not necessarily prepared him for a determining role in the methodology of his own case. For one thing, the increasing contribution of personal habits and life styles to the ill and injured population does not invite the conviction that the patient's medical judgment qualifies him for a major degree of control. We would personally accept the formation of a lay committee to dictate the conduct of medical practice with one provision—that at any meeting at which policies were to be established, at least half of the members should be ill or in pain. Now *that* would provide the perspective under which a worthy medical policy could be established.

Physicians who find their way onto such panels seem to wind up in a patterned response. They agree with their co-panelists regarding the culpability of the profession generally (while assuring that in their own offices they have always locked the patient into the decision-making circuit) but everything is changing, just you wait and see. (This is a pattern characteristic of the medical debater when confronted with some alleged shortcoming: it isn't so and anyway we're doing better). But the individual physician (who may or may not feel properly represented by his impaneled colleague) functions from an awareness that many of his patients still want to rely on his advice and direction and that much that is advocated has either been present in a less visible form, or represents window dressing which will have little value in the medical service, or it is a mechanism for transmitting this control not to the patient but to the promoting organization. The panelists deal, of course, in generalized figures in citing the need for change—"many patients"—"large numbers"—"the public" is demanding change. Statistics, if they have them, are suspect and by their nature constitute a bias of ascertainment, contributing more to headline than scientific assessment.

So the basic conflict in these exchanges seems to be the discrepancy between the generalized concept of service provided by one group to another group as interpreted by a third group on the one hand, and the personalized concept of the individual physician serving an individual patient on the other. Ironically, in purporting to speak for this supposedly disfranchised person and demanding a voice for him in his medical management, these advocates lump them into one homogeneous mass, supposedly of one mind, and that being

what the advocates say it is. They cite large numbers of individuals as evidence of the dissatisfaction when any knowledge of human nature testifies that the larger the group the more diversity there is to its opinions. The physician, then, extrapolates an interpretation of what the public wants from his dealings with individual patients, while the consumerist claims to know the individual needs from his general observation.

The medical profession has a penchant, when confronted with problems of this sort, for appearing to deny that any problem exists, or pursuing a self-serving avoidance of social responsibility. Neither charge is fully justified but the fact of their appearance expresses the need for a continuing effort to develop a better working relationship between physician and patient. No amount of denial on the part of the profession will nullify the fact that medical practice has changed and, even if the basic physician-patient relationship is still there, the character of the exchange of feelings between these two principals has changed. The physician has tended to let the technology speak for him, to assume that the wonder of medicine's complexities and accomplishments should impress the public in a manner that called for acceptance without personal interpretation. The public has responded by demanding more and more while understanding less and less—and everyone wonders why the two can't talk together as they once did.

We are, as you see, back to the old problem of communication and the new scene, far from letting physicians off easy, is more demanding of them to interpret their functions effectively. Still, better communication will serve both better than just giving the patient the vote. We think the patient doesn't need or want the burden of all the facts and options. We think he still wants to rely on the opinion and guidance of his physician, but he wants to feel it is the opinion of his physician, not the clicketyclacking machine; that it is the recommendation of his physician, not the computer-dictated set of directions relayed by an office assistant. He doesn't necessarily want control but he wants a grip on the person who is in control.

This problem of communication obviously isn't new. Its maintenance isn't easy and it does need overhauling occasionally. It has a way of getting lost at times and turning up intermittently under the guise of some group insistence on a change of method. The fact is that most of the attitudes expressed by the pressure groups are not without merit—at least deserving of an objective assessment. The conflicts which arise represent a mechanism of social accommodation by which institutions move from the needs of one day to those of the next. But in the process it becomes necessary for the medical profession to insist upon appropriate consideration for those elements that have served well in the past.

So we contend that not every patient need be given every item of information nor, if he wants to gain optimum benefit from his physician's services, expect to pick and choose what he will take of the services. But in general, physicians probably underestimate the patient's ability to handle the difficult things. This is a remnant of the day when the physician's capabilities and options were much more limited. The care he provided was primarily an authoritative communication—owing its authority as much to its manner of delivery as to any scientific content. Patients weren't always content—when in history has the physician been free from critics?—but their discontent was a personal matter, not a *cause celebre* calling for mobilization of a group dedicated to reforming the profession. One might suppose that the undeniably successful record of medical care would bring a degree of contentment to the public. It would seem that the patient's medical sophistication, a by-product in itself of this record, would foster better understanding of the need to rely on the authority of the physician. The detractors, by the volume and intensity of their alarums, indict the whole profession. The current climate is determined by the expectation of unfailing success, the conviction that illness is an unjustified intrusion upon the individual for which someone (else) should be accountable, the social promise that everyone shall have everything—and has led to the conclusion that only by giving the patient control can the medical service be properly provided.

Perhaps the physician should accede to this demand for the patient-physician relationship in renovated form. It may resolve a couple of other problems. Since there is a demand for second opinions, why not let the patient provide it? And if he contemplates a malpractice suit, perhaps he will be in the interesting position of having to include himself among the defendants.

We are not overlooking the fact that some physicians, by certain innate personality features, by lack of time, by preoccupation with things that seem more important, or whatever, do not translate medical information and qualifications into terms the patient can grasp and act knowledgeably upon (which is what he really wants). There are such and there is probably no magical way to change them. But the medical profession must maintain control over the medical function just as the individual physician must maintain control over the individual patient. And this can be done only when the patient feels included in the process not just as an illness but as a person. With this assurance provided by his physician's management and attitude, he will gladly turn his proxy over to the physician. It should be an inviting prospect for the physician, for insofar as this is accomplished the profession will be free of the planners who feel they must make the rules.—*D.E.G.*

Guest Editorial

Physician Manpower in Kansas

JOHN RAND NEUENSCHWANDER, M.D., ***Hoxie***

(Editor's Note: The following remarks were prepared by John Rand Neuenschwander, M.D., for presentation at the Physician Manpower Symposium held in Topeka, November 1977. THE JOURNAL is pleased to publish it as an overview of the medical manpower situation particularly in the rural areas, and as experienced by a second generation family physician.)

I AM THE SON of a physician who has practiced for 20 of the last 25 years as the only medical doctor in Hoxie, Kansas, population 1,500, and Sheridan County, population 4,000. As a child, I saw rural physicians on call 24 hours a day for five years without vacation; I saw some die at age 40 from the stresses of practice.

I was graduated from the University of Kansas Medical School in 1972, served an internship at Bethany Hospital in Kansas City, and completed a Family Practice Residency in Cedar Rapids, Iowa. All family practice residents were acutely aware of the medical needs of rural areas and our potential roles in meeting them. I returned to Kansas and for the last three years have been in practice in Hoxie with my father.

I have seen the medical manpower problems of rural Kansas from the perspectives of physician's son, patient, medical student, resident, and family physician. It is from this background that I discuss the physician manpower shortage in Kansas.

The Problem

What is the medical manpower problem in Kansas? Simply, it is that there is an insufficient number of physicians to care for the population, and distribution is such that some areas are comfortable while others are desperate for care.

However, the situation is seriously exaggerated. A newspaper article on the physician shortage, which boasted accurate and current data, listed my county as desperately underserved with one physician, when in fact we have two MDs and one DO; similar errors existed in the figures for surrounding counties. Rural citizens are constantly told they are underserved, and most would be amazed to find that they spend less time obtaining medical care than their urban counterparts—if I should suffer severe injury or illness, I would prefer to be in western Kansas. However, many areas do need more medical manpower, and we are addressing this problem today. What are the factors involved?

Medical Education

There has been recent concern over medical school admissions policy. An often quoted study indicates that physicians rarely settle in a community smaller than their home town. This statistic is distorted because it was gathered at a time when nearly all physicians were specializing and could not possibly settle in a rural area. Many young family doctors are now seeking smaller communities, for reasons I will explain later. Still, origin is a factor. Although the number of rural applicants accepted to medical school appears small, the percentage accepted, rural versus urban, is comparable. Added to this are those rural products who are accepted with current addresses of Lawrence or Manhattan. There needs to be an increase in the number of qualified persons applying from rural areas. There is no easy way to do this, but the recent expression of interest from KUMC in rural students and publicity regarding rural health care will help.

The student's best exposure to primary care is the precepteeship, where he spends a month sharing the professional and social life of a rural physician. My father has been a preceptor in this program for 20 years. His experience, as well as KU surveys, show that students consider this rotation among the best, and nearly all are favorably impressed with family practice, regardless of their field of interest. About 1971, KUMC allowed the precepteeship to be in specialties and cities, such as Dermatology in Wichita. As a result, more graduates maintained erroneous concepts of rural life and practice. A poll of members of the Northwest Kansas Medical Society (NWKMA), of which I am a member, revealed that 85 per cent of the membership felt that it would be helpful for KUMC to return their precepteeship to a rural primary care experience.

The greatest factor in a physician's choice of career is societal attitude. Ten years ago the family physician

was universally considered a second-class doctor. It was a status symbol to have all your care provided by specialists. Family doctors were considered by many to be those who lacked the abilities or determination to further their education. Such attitudes were and are wrong, but it is not surprising that medical students of those times shunned family practice. Now the family physician is esteemed—every family is proud to have their doctor provide or guide their total health care—and now so many young doctors want family practice training that residencies are overfilled, the only residencies in the United States with more demand than supply. This has all happened without legislative intervention. I vividly remember my first day of medical school—my class was asked how many wanted to be family doctors, and less than 15 per cent raised their hands; at graduation fewer than 10 per cent responded. That question would elicit positive response from half the students in today's classes. Some critics curse the medical school for not producing 50 per cent family physicians from my class, conveniently ignoring the fact that 85 per cent of the students had no interest in family practice when they entered. A state representative, in his column in my hometown paper, said, "We are serving notice to the Medical Center that the legislature expects them to train doctors for rural Kansas, or future state support will be reduced or terminated." Such an attitude is not only naive but counterproductive, and should be condemned.

Family Practice Residencies

The main component of the solution to the rural physician shortage is the family practice residency. It was born of the desire of physicians to enter a general practice with education, experience, and skills suitable to the needs of their patients. The residency helps create a physician ideally prepared for a rural practice. Although 90 per cent of the members of NWKMS completed no residency, 80 per cent feel that any physician seeking a rural practice should complete a primary care residency. It is ironic that this affirmation of the need for superior knowledge and skill in a rural practice should come at a time when Physician Assistants are being considered to replace physicians in some communities, and many legislators are voicing more support for physicians who do not complete residencies. These residencies help prevent the sense of insecurity which drives physicians to urban practices surrounded by specialists, and provide competence in skills which the physician can ideally use in a rural practice. Family physicians are finding that city hospitals deny them the privileges of doing tonsillectomies, delivering babies and caring for heart attacks, even when they can document training and skills in these areas. They find that their skills best correspond with the needs of a rural practice. One of my fellow residents, Dr. Ken Olson, surveyed family practice residents in the midwestern United States and found that 16 per cent sought to practice in communities with populations under 5,000, and 54 per cent in areas with fewer than 10,000. The American Academy of Family Physicians made a similar study of all 1976 United States graduates and found that 16 per cent sought areas of practice with populations of 5,000 and less, 37 per cent chose 15,000 or less. This trend evolved without coercion or penalties; it is a simple result of recognition by society of the needs and encouragement toward solutions.

Physicians and educators feel that this trend toward rural medicine will provide adequate manpower over the next several years. Predictions of massive shortages are based on the questionable figure of one family doctor for every 700-1,000 persons, and all family doctors I know consider that figure ridiculous; a ratio of 1 : 2,000 or 2,500 is more reasonable. A physician must be busy to maintain low fees and adequate knowledge. The federal government recognized the importance of primary care residencies, and was planning to mandate quotas to attain 35 per cent primary care residencies. That figure has already been met.

Rural Kansas

Small towns have much to offer the Family Physician. They possess the world's nicest people, and serving them is much more rewarding and less difficult. The environment is nearly free of crime, pollution and rat race. Here is the last frontier, where physician is friend rather than adversary, and people are proud and supportive of their health care facilities.

Many areas have excellent small hospitals. Hoxie has a 19-bed hospital and a 38-bed long term care unit. We have physical therapy, cardiac monitors, and lab facilities including telemetry EKG, blood gasses, and Hycel profile; we are obtaining fetal monitor capabilities. Our hospital has had Joint Commission approval yearly since 1968—the smallest hospital in Kansas and possibly in the United States to be so honored. Yet the government seems determined to destroy this facility. Standards designed and appropriate for 500-bed institutions are applied more vigorously and punitively to small hospitals. Payments are insulting; although our hospital's charges are below city levels, it receives only half payment from Medicare. The average length of stay for most disease states is well below the big hospital average, testifying to our hospital's efficiency. Its services are indispensible to the elderly, half of whom live in rural areas in Kansas.

Any community without good hospital facilities is very unlikely to attract or keep a physician; I am certain that if Hoxie loses its hospital, it will lose its physicians, and most physicians in this area share this belief. This is not a threat, it is a statement of fact. It is not negotiable. The hospital provides the environment for the physician to handle complex cases, obtain special lab work, care for emergency patients, and generally keep current in medicine. It is very difficult to provide good medical care without a hospital; if a community does not allow the physician to provide quality care, he will leave.

Scholarships

In northwest Kansas, the Dane Hansen Memorial Fund provides scholarships to students in health fields who promise to return to northwest Kansas. The legislature tried a similar program one year, had little student acceptance, and abandoned it. That year many Hansen scholarships also went unaccepted, and the selection committee agreed that although this gesture of good will and support for rural health practitioners was worth while, one cannot buy physicians or dentists for rural Kansas. Recently, as predicted two years ago, the Hansen Fund has had more applicants than it can handle. Perhaps the legislature will wish to try again, although they can only reward, not coerce, a student's decision toward rural practice.

Admission Quotas

As rural students are thought more likely to return to rural areas, a medical school admission quota has been suggested. The most frightening proposal would allow appointment of students from senatorial districts. Obviously, any admission based on criteria other than ability and merit will adversely affect the excellent quality of graduates for which KUMC is justifiably famous. Furthermore, the graduating family physician does not need the stigma of being selected and trained for political expedience rather than accomplishment. Eighty per cent of the membership of NWKMS oppose any concept of area quotas, but all feel it would be helpful for KUMC to increase its recruitment activities in rural areas. Thankfully, the recent surge of interest in family practice makes any quota unlikely.

Conscription

One of the more controversial suggestions has been a financial penalty for medical students. Most recently this has been described as increasing KUMC tuition from among the lowest to among the highest in the United States, and then forgiving part of the tuition for graduates who practice in deprived areas. Described in this manner, the proposal will sound reasonable to many. Unfortunately, the damage has been done—the proponents of this measure made it quite clear during its formation that it was intended to be punitive. One representative stated on television, "It is time that medical students started paying back the state for their education, either in money or in services." This is a grim precedent in Kansas education. Original estimates set the graduating student's debt at $80,000 to $100,000—more if he took a residency in Kansas. This has led many students to believe that rural Kansas is so dismal that only through conscription can necessary services be obtained; and since proponents of this measure claim massive public support, students begin to see the people of rural Kansas with alarm and contempt rather than sympathy and concern. In my experience, no such public support exists, and the concept of governmental coercion in one's occupation is incompatible with the principles of rural people. If this program were adopted now, it could not possibly produce results for five to seven years, and is certain to be inappropriate by that time. Ninety per cent of the physicians in NWKMS strongly oppose this bill, many flatly stating that they want no help given under duress or on a temporary basis. Medical educators universally agree that this measure would adversely affect the quality of student our schools would attract; many plan to resign if this bill passes. This concept of conscription has caused enough ill will already; it should be abandoned.

Equal Pay for Equal Services

When I entered practice, I could set my own fees, but Medicare, Medicaid, and Blue Shield now tell me what I will be paid. As I did not wish to set fees that would penalize the patients who pay their own way, my fees were in essence set by these groups. They will pay my city colleagues twice as much. Let me state that the wolf is not at my door—I am not concerned over my ability to make a living. We should all be concerned, however, that our government would officially state that when one serves a rural population, his services are worth only half as much. (I made this statement based on personal experiences with Medicare and Medicaid, and the opinions of my colleagues in rural practices. Dr. Fitch, of the Medical Services Section of Kansas SRS, has stated that no statutory or intentional pay differential exists. I am now convinced that he is correct, and have sent him a letter of apology. However, it is significant that many physicians feel there is such a differential, or at least that there is no practical way for a rural physician to be reimbursed at urban levels. Furthermore, an error

was made in my specific case in which Medicaid fixed my office fee at $5 when I began practice in 1975, and it remains frozen there. In my letter to Dr. Fitch I requested correction of this error and was told, ". . . we can do nothing about it due to the federal regulations with which we must comply. . . ." To state that Medicare and Medicaid officially discriminate against the rural physician is not true—the distance to the truth, however, is inconsequential.)

Although my county has a small welfare population, I have spoken with physicians whose practices consist mostly of Medicare or Medicaid patients. Some collect only ten cents on the dollar, and the state's policy of partial, delayed, and refused payment does them serious hardship and injustice. It is ironic that with all the imaginative plans to penalize, reward or manipulate physicians, the legislature has ignored the simple tradition of equal pay for equal service. This concept is supported by 85 per cent of the membership of NWKMS.

Considerations

Rural Communities

The small community and the economy car both existed ten years ago and both were largely ignored, but times have changed. At this time, unprecedented numbers of physicians in training are planning rural family practices. Communities can make their opportunities known to residencies and medical schools, and through organized meetings of students and community representatives, such as Kansas Health Day.

Promises of loans, guaranteed incomes, or low cost facilities are useful primarily as gestures of good will. Rural physicians are aware that they could double their incomes in urban practices.

A physician is unlikely to settle in a community which cannot offer the support of a local hospital, proper lab facilities, adequate nursing and paramedical personnel, and other physicians to share responsibilities and problems. The day of the solo physician in an isolated office is past.

Rural youth should be encouraged to seek careers in medicine. At this time, their background will be a favorable influence on admissions committees.

Medical Schools

Consideration should be given to an applicant's background, origin, and practice intentions when selecting students. Strict quotas should not be used.

Students should be exposed to family practice and primary care physicians during their education through such programs as precepteeship.

Support and encouragement should be extended to family practice residencies. Care must be exercised to assure that residents have productive experiences in surgery, ob/gyn, pediatrics, and other rotations. If the resident is continuously a low teaching priority at large institutions, better educational opportunities should be found elsewhere.

Kansas Legislature

With the resurgence of interest in Family Practice and the recent increase in physicians seeking rural practice, little legislation is needed, and radical legislation is likely to be counterproductive.

Scholarships to physicians seeking practices in deprived areas may help, but will tend to reward rather than influence their decisions.

As Family Practice residencies are the key to solving the Kansas physician shortage, they must be adequately supported. Residencies based in smaller institutions, with rural exposure and few competing specialty programs, have better reputations for quality primary care education than do programs in large urban teaching institutions. There are indications that residents are likely to practice near their training programs. A Family Practice residency in western Kansas should be ideal.

Medicare, Medicaid, and insurance companies should be encouraged to cease payment practices that punish physicians who serve rural or deprived areas, and needy or elderly populations.

The plan to make medical education expensive to those who resist legislative dicta has already driven wedges between medical students and medically underserved communities, and it should be abandoned. Conscientious, concerned health care cannot be obtained through coercion.

The heated political rhetoric surrounding Kansas health care has had many unfortunate consequences. We are told through the press that KUMC is insensitive and self-serving, and therefore requires legislative control; that medical students are lazy, ungrateful, and respond best to threats or bribery; that physicians consider profit more important than facilities, community, or practice opportunities. The traditional warm doctor-patient relationship is suffering under this attack. The health needs of Kansas will be better met through a spirit of cooperation and understanding than through animosity and mud-slinging. The legislature's success in improving health care in Kansas will not be measured by the number of radical bills hastily passed.

A Medical Metaphor

There is no cancer of our health care distribution system. It has a flu—possibly a swine flu—and shows all indications of gradual recovery through its own natural

defenses. Physicians, relatives, and friends have all expressed concern; their gentle support is appreciated. The learned physician knows that some illnesses cannot be boldly cured, and that radical therapy may be of greater risk than careful observation and supportive measures.

There are those among us, however, who insist that a swift and glamorous cure must be found; that the patient cannot improve without their grand intervention. These are the quacks. Their desire to gain profit or fame at the patient's expense is contemptible. Had their suggestions been accepted two years ago, they would now be claiming credit for the patient's improved condition.

In seeking solutions to our health care problems, let us be guided by the traditional principle of medicine:

Primum Non Nocere
(First Do No Harm)

Drug Regulation

(Continued from page 306)

If a drug appears to be a major breakthrough, FDA could approve it provisionally while further research to confirm its effectiveness is being conducted. Provisional approval expires in three years unless renewed. (Currently FDA has no authority to approve a promising drug provisionally until all tests are complete.)

In exceptional cases where a drug could not be approved for general marketing, FDA could approve it with special distribution requirements. Thus, drugs that could not be available under the present regulatory system because their risks for most patients may be too great, could be approved with distribution and dispensing restrictions for those few patients who might benefit from them.

After drug approval, the bill would require routine surveillance of the drug by the manufacturer unless FDA finds it unnecessary. FDA also could require manufacturers to conduct post-approval studies, if necessary, to detect unexpected adverse effects, to determine how the drug should be used in special patient groups such as children, or to see whether the drug is effective for other purposes. (There is no current provision clearly authorizing FDA to require post-approval surveillance or additional studies.)

If, after approval, FDA believes a drug presents an unreasonable and substantial risk of harm, it could quickly suspend approval. The legislation allows the Secretary of HEW to delegate this authority to FDA. (Currently, only the Secretary of HEW can remove a drug from the market immediately, and only if it poses an "imminent hazard.")

Yeast Vaginitis

(Continued from page 323)

both forms of medication were equivalent with regard to relief of the symptoms by the 14th or 15th day after inception of therapy. Likewise, the rate of relief was essentially identical for the cream and suppository groups, as indicated by the individual results. The symptoms of discharge and odor were not included in the analysis since both medications have a tendency to obscure these evaluations.

Of the 26 patients who completed the series in the group receiving the cream, 22 (84.6%) were clinically cured, 2 (7.7%) failed to respond to therapy, and 2 (7.7%) received only symptomatic relief. Of the 28 patients completing the series using the suppositories, 24 (85.7%) were clinically cured, 1 (3.6%) failed to respond to therapy, and 3 (10.7%) received only symptomatic relief.

In the group utilizing the cream therapy, one patient reported stinging and itching while using the medication. Many patients noted that the cream was messy and the pad worn to prevent rapid discharge of the medication would often irritate the vulva, thus adding to their discomfort. Of the patients receiving the suppository treatment, two noted burning, one noted itching and burning, and one noted a medication odor. In neither treatment group was discontinuation of therapy required due to the above-noted complaints. One patient using the suppositories reported severe irritation which subsided after she voluntarily discontinued the medication on the 11th day. This patient was found to be a clinical failure at the completion of the series.

Summary

Both groups of patients entered in this study were essentially equivalent as to demography, the initial complaints, and the initial assessment of clinical signs from the monilial infection. The rate of relief of itching, irritation and burning symptoms, as recorded by the individual patients, was also identical. Clinical evaluation one month after completion of therapy supported equivalence by revealing that the regimen was a success in 84.6 per cent of the patients receiving cream and in 85.7 per cent of those receiving suppositories. Patients evaluated as failures or symptomatically relieved were found in 15.4 per cent of those receiving cream and 14.3 per cent of those receiving suppositories. The suppositories were well accepted by the patients, and were as safe and effective in the treatment of monilial vulvovaginitis as the cream.

Drugs, Diet, and Nutrition

Whereas the intake, absorption and metabolism of nutrients may be adversely affected by the long-term intake of certain therapeutic drugs, it is also true that the absorption and metabolism of drugs is affected by the diet. Recent research suggests that we need to be concerned with these diet-drug interactions. With increases in the range of medications available to patients and the widespread and rational use of drug therapy in the control of chronic disease, it is necessary to identify risks of nutritional side effects. Further, we need to know that the predicted and intended therapeutic effects of drugs are not diminished or otherwise altered by diet or change in diet.

Effects of Drugs on Appetite and Food Intake

Drugs can reduce food intake because they cause a perversion or loss of appetite; because they induce sedation; or because they evoke an adverse response when food is taken. Drugs affecting appetite may do so as a central or peripheral effect. Appetite reducing drugs, acting centrally, include unsubstituted and substituted amphetamines as well as related compounds. Whereas this class of drugs has not shown outstanding effectiveness in the treatment or control of obesity, it has been noted that in children given such stimulant drugs for the control of hyperactivity, growth retardation may occur while the drug therapy is maintained. Over-the-counter weight reducing drugs, which may contain bulking agents such as methycellulose, and mild stimulants such as phenylpropanolamine, an ephedrine-like drug, have not been shown to have a marked effect on food intake or on obesity.

Drugs which cause nausea will tend to reduce food intake and contribute to weight loss. Digitalis, given for long periods of time at high dosage, may cause severe wasting in some patients, because they are not inclined to eat and suffer nausea. Cancer chemotherapeutic agents can induce loss of appetite, both because they may cause nausea and vomiting, and also because of inflammatory reaction within the upper gastrointestinal tract. Poor appetite induced by drugs is a more severe problem in patients who are already wasted because of the underlying disease for which the drug has been prescribed. Drugs may have different effects on appetite at different times or under different circumstances. For example, phenothiazine tranquilizers tend to increase appetite in psychotic patients, both as a direct drug effect and also because mental agitation is reduced. However, if these same drugs are given to elderly patients so that the level of consciousness is depressed, then food intake may also be reduced. It is important to realize that in patients who are on a marginal diet, *i.e.*, marginal with respect to required nutrients, appetite reducing drugs can contribute significantly to the development of nutritional deficiencies, including vitamin deficiencies. This was documented in elderly patients in extended care facilities who are on multiple drug regimens.

Certain drugs, including cyproheptadine (Periactin), can increase appetite. This effect has been utilized in the nutritional rehabilitation of debilitated persons.

Drugs and Nutrient Absorption

Drugs in common use which can cause nutrient malabsorption include antacids, laxatives, including mineral oil and phenolphthalein, the antibiotic neomycin and colchicine, which is used in the treatment of gout, as well as cholestyramine, currently being used to reduce serum cholesterol in patients with atherosclerotic heart disease. Abuse of antacids can lead to phosphate depletion and in some patients to the development of osteomalacia (vitamin D deficiency). Mineral oil diminishes absorption of fat-soluble vitamins and carotene. Abuse of mineral oil may lead to rickets in the child and osteomalacia in the adult. Abuse of laxatives containing phenolphthalein can cause a malabsorption syndrome with the development of multiple nutrient deficiencies as well as excessive loss of potassium from the body leading to hypokalemia and protein-losing enteropathy (excessive loss of proteins into the intestine). Neomycin, an antibiotic used to "sterilize" the large intestine and to prevent the development of hepatic encephalopathy (a brain syndrome) in alcoholics with advanced liver disease, has long been known to cause malabsorption. However, the medical justifications for the use of the drug are such that the risk of malabsorption may be justified. Colchicine, which is used in the treatment of gout, will rapidly evoke malabsorption associated with the development of diarrhea. Since this drug is usually only given for the treatment of acute gout, its adverse effects on nutrient absorptive processes are rarely such as to require drug withdrawal.

Studies of malabsorption induced by cholestyramine require careful evaluation at the present time because this drug may be prescribed as a long-term therapy. In children, it has been shown that depletion of the B vitamin, folic acid, may result from continued intake of cholestyramine. This drug also reduced absorption of fat soluble vitamins, notably vitamins A, D, and K.

Effects of Drugs on Nutrient Metabolism

Under certain conditions, the desired therapeutic effect of a drug is by deliberate interference with the body's utilization of a particular nutrient. Methotrexate, a drug of choice in certain types of leukemia and in cancer, prevents or interferes with the metabolism of folic acid. Anticoagulants used to treat or prevent thrombophlebitis are, in fact, antivitamin K compounds. With other drugs, anti-nutrient properties are recognized as an avoidable side effect. Isoniazid and several other drugs used in the treatment of tuberculosis as well as the drug L-dopa, used in the treatment of Parkinson's disease, inhibit the normal metabolism of vitamin B_6.

Anticonvulsant drugs, which can also be anorectic, may alter the metabolism of several nutrients including vitamins D and K. These same drugs, more especially diphenylhydantoin and phenobarbital, cannot only induce a deficiency of these vitamins with prolonged usage but can also cause a folic acid deficiency. In documented cases of vitamin D deficiency and folic acid deficiency associated with intake of anticonvulsant drugs, effects have been shown to be due to decreased intake or metabolism of these vitamins in the bodies of patients already consuming a marginal diet. Prenatal or perinatal malnutrition can be induced by drugs. For example, neonatal vitamin K deficiency

can arise in the infant of an epileptic woman who has been on anticonvulsant drugs during pregnancy. There is some concern that prenatal folic acid deficiency could be induced by anticonvulsant drugs, causing interference with the development of the fetus. Whereas the risk to the fetus from uncontrolled epilepsy is high, there is no clear evidence that folic acid deficiency induced by anticonvulsant drugs in the pregnant woman can lead to specific adverse effects on fetal development. While multiple fetal abnormalities (the fetal hydantoin syndrome) have been associated with intake of diphenylhydantoin during early gestation, there is no definitive evidence that this syndrome is related in any way to fetal deficiencies either of folic acid or of vitamin D.

High doses of folic acid given to epileptics on anticonvulsants may decrease the effectiveness of the therapeutic agents by lowering blood levels of diphenylhydantoin. A similar situation can occur if high doses of folic acid are given to such patients who are on phenobarbital. Therefore, in considering the prevention or treatment of folic acid deficiency in epileptics, it is necessary to weigh these considerations and only to give folic acid in physiological amounts.

Nutrition and the Pill

Nutrient requirements may be increased in women on oral contraceptives. While oral contraceptives have not been reported to cause overt nutritional deficiency except in a few patients who have been on deficient diets or who have malabsorption syndromes independent of the Pill, blood levels of several nutrients are altered by the intake of these agents. In particular, it has been shown that patients on the Pill for long periods of time may develop B vitamin depletion including depletion of folic acid, riboflavin and vitamin B_6, as well as vitamin C. Blood levels of other nutrients are increased by intake of the Pill, including vitamin A. It is important to reiterate that women on the Pill are only at risk for the development of vitamin deficiencies if their diets are grossly lacking in these nutrients and, more particularly, lacking in folic acid. Prevention of nutrient depletion in Pill users requires identification of women at risk, diet counselling, suggested intake of foods fortified with these vitamins and if these measures are inadequate, prescription of physiological vitamin preparations formulated to the needs of women on the Pill.

Effects of Food on Drug Absorption

Absorption of drugs may be increased or decreased by feeding, by foods, by specific nutrients contained in these foods, or by non-nutrient components of the diet. Delayed absorption of the following drugs occurs when they are taken at the same time as food: amoxicillin, cephalexin, sulfanilamide, sulfadiazine, sulfamethoxine, sulfisoxazole, aspirin, acetaminophen, digoxin, and furosemide. In the case of acetaminophen, the food effect appears to be due to the pectin content of the diet. Reduced absorption of drugs may also be due to eating at the same time as the drug is taken. Investigations are required to define which specific components of the diet affect drug absorption. Our own concern is with dietary fiber sources, because it is generally considered desirable to increase dietary fiber intake in order to avoid constipation and other bowel disease such as diverticulosis. There is a need, therefore, to define whether specific dietary fiber sources can slow or impede drug absorption, since this may alter the time at which patients attain desired blood levels of specific drugs intended for the treatment of specific infective or other diseases.

Diet and Drug Metabolism

In animal as well as human studies, it has been shown that changes in the diet may affect the rate of drug metabolism. Certain drugs are metabolized faster when a patient is on a high protein-low carbohydrate diet rather than a high carbohydrate-low protein diet. Similarly, specific non-nutrient dietary components may speed up drug metabolism, including compounds present in cabbage and brussels sprouts. Cooking methods may alter the rate of drug metabolism, particularly charcoal broiling, which may promote conversion of drugs to their metabolites. Changes in the diet which alter the range of bacteria in the gastrointestinal tract may also alter the rate of drug metabolism or the range of metabolites produced. Effects on the gut microflora are produced by changing dietary fiber sources and levels as well as by changing the level of animal protein in the diet.

Who Is at Risk?

Drug-induced nutritional deficiencies are likely to occur in long-term drug users who are already at nutritional risk because of a defective diet, chronic disease, alcohol abuse, or physiological stress. Patients in whom such deficiencies have most frequently been identified include drug abusers such as laxative abusers, the elderly, particularly those in extended care facilities receiving several drugs which can affect nutritional status, in alcoholics, and in children during the period of active growth. Drug-induced malnutrition is largely preventable. It is necessary that physicians be made aware of this drug risk; that nutritional deficiencies present are precisely identified by laboratory studies; and that dietary changes with nutrient supplementation be prescribed. Nutrient requirements of persons receiving certain drugs over long periods of time are different from those of people not on these drugs but, unfortunately, this fact is not yet generally appreciated.

Since therapeutic diets are frequently prescribed for patients receiving drugs at the same time, it is necessary to know what drug-diet interactions are likely to occur. Further, in order to optimize the therapeutic effects of drugs and to minimize side effects, consideration must be given to the optimum time for drug administration, whether this should be with meals or between meals. Accurate writing of prescriptions and labeling of drug bottles would serve largely to obviate the misunderstanding on the part of the patient as to when the drug is best taken. If these precautions are not carried out, then it is likely that many therapeutic drugs may not be optimally absorbed. Change in the rate of drug metabolism with change in diet is now beginning to be predictable. Further research, however, is required to identify the various effects of change in diet composition on drug metabolism.

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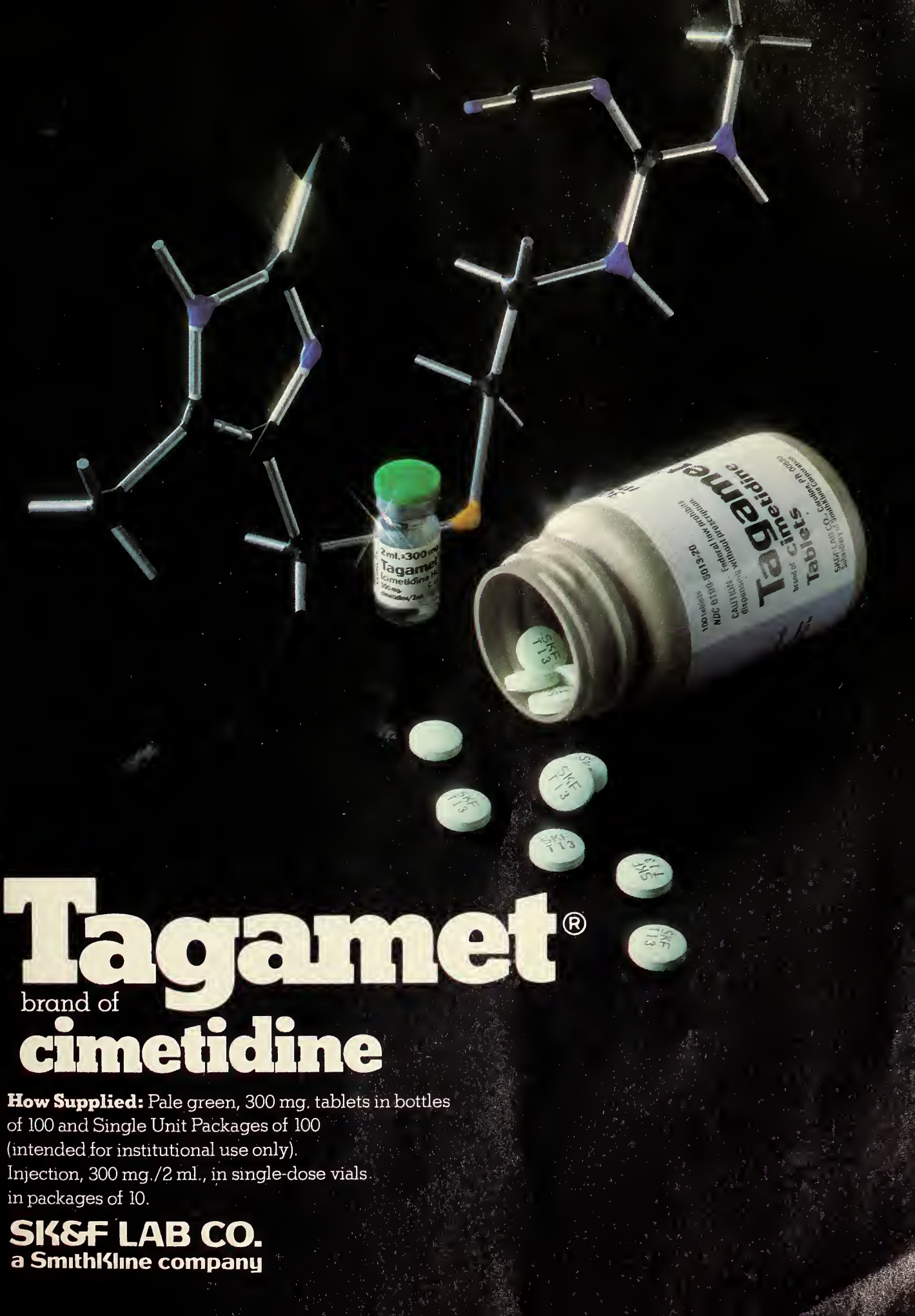
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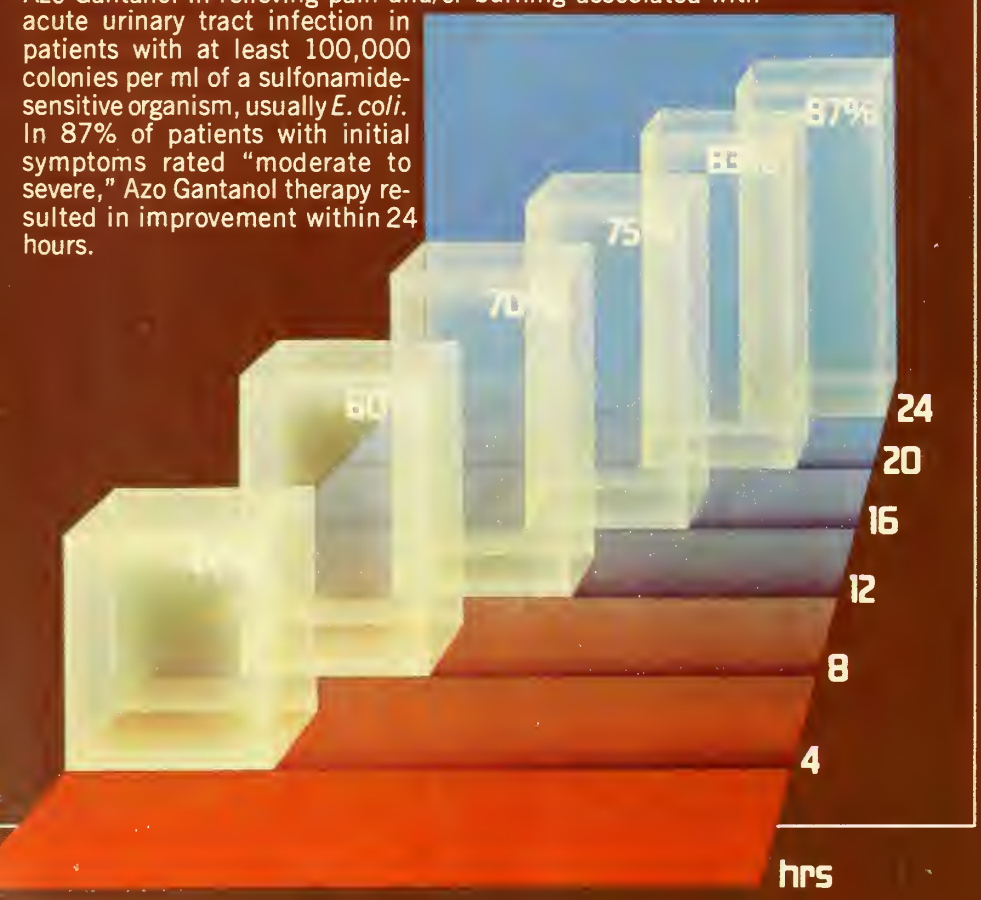
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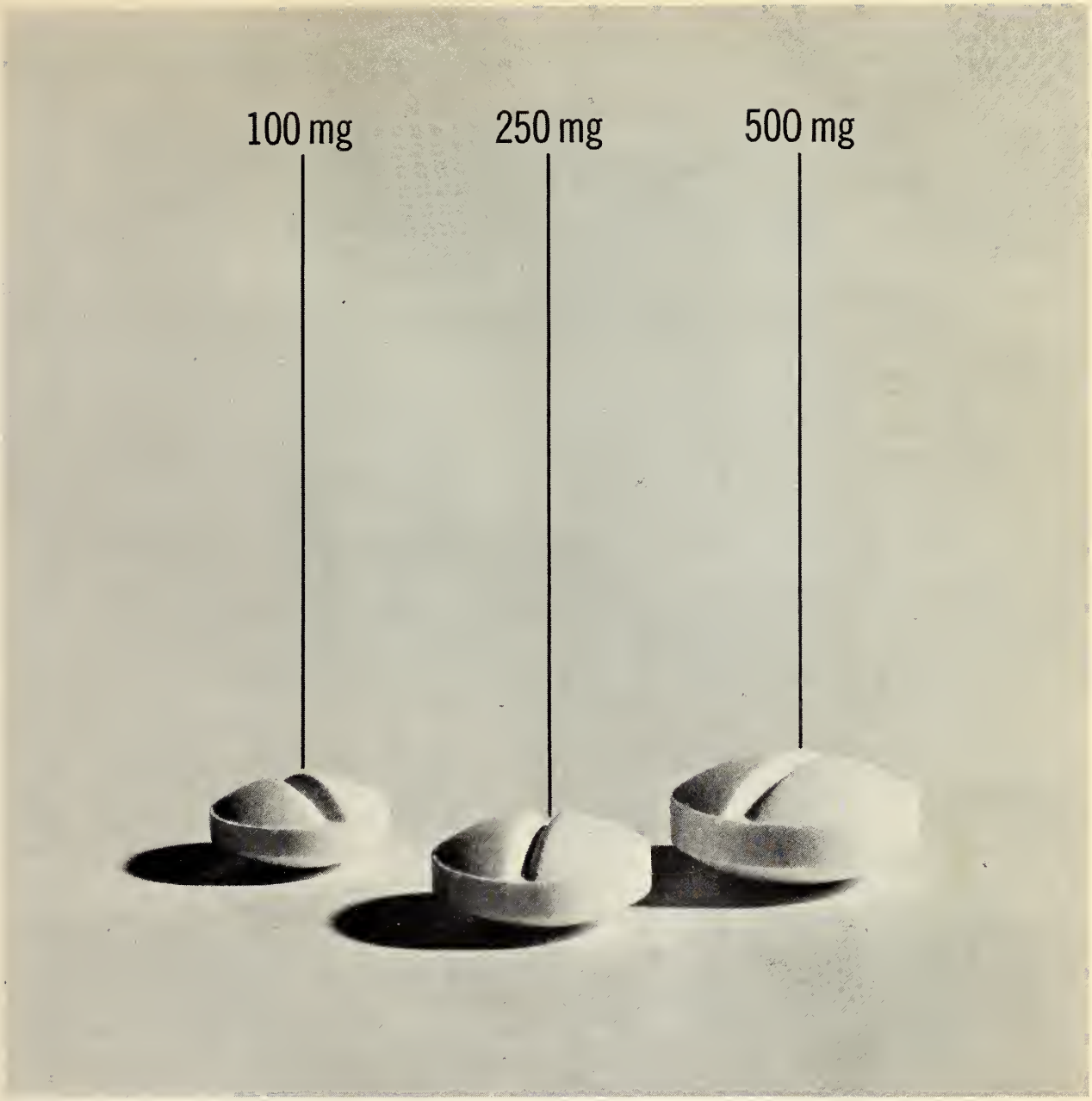
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Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms (similar to those with barbiturates, alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal/muscle cramps, vomiting, sweating). Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL: Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral form adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

INJECTABLE: *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling, and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins, i.e., dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

Administer with extreme care to elderly, very ill, those with limited pulmonary reserve because of possibility of apnea and/or cardiac arrest; concomitant use of barbiturates, alcohol or other CNS depressants increases depression with increased risk of apnea; have resuscitative facilities available. When used with narcotic analgesic eliminate or reduce narcotic dosage at least 1/3, administer in small increments. Should not be administered to patients in shock, coma, acute alcoholic intoxication with depression of vital signs.

Has precipitated tonic status epilepticus in patients treated for petit mal status or petit mal variant status.

Withdrawal symptoms (similar to those with barbiturates, alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal/muscle cramps, vomiting, sweating). Keep addiction-prone individuals under careful surveillance because of predisposition to habituation/dependence. Not recommended for OB use.

Efficacy/safety not established in neonates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of Valium (diazepam), *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed or tolerated).

INJECTABLE: Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, have been observed in patients during and after Valium (diazepam) therapy and are of no known significance.

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In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension, caffeine and sodium benzoate for CNS-depressive effects. Dialysis is of limited value.

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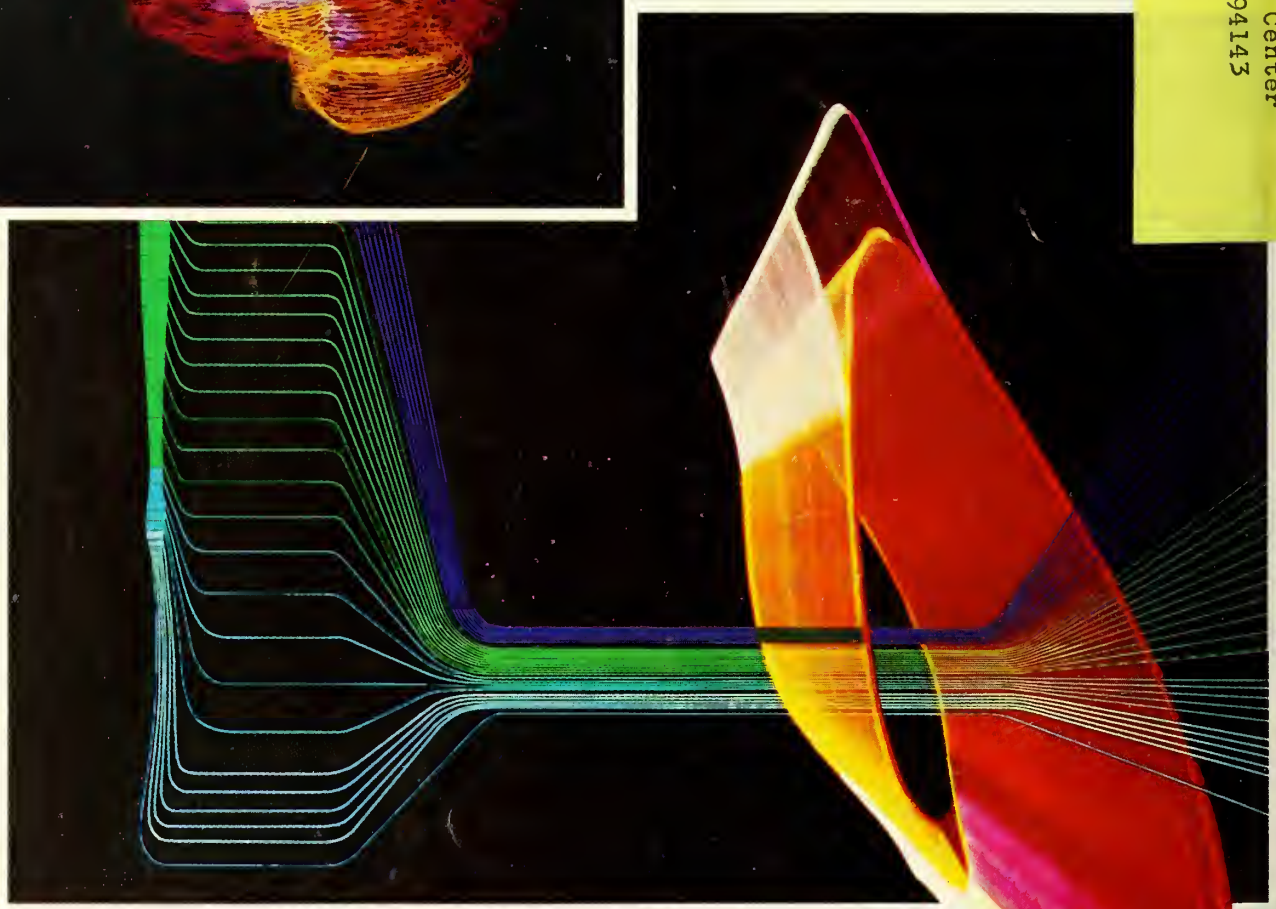
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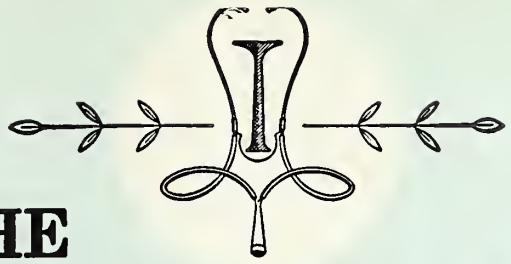
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The JOURNAL of the KANSAS MEDICAL SOCIETY

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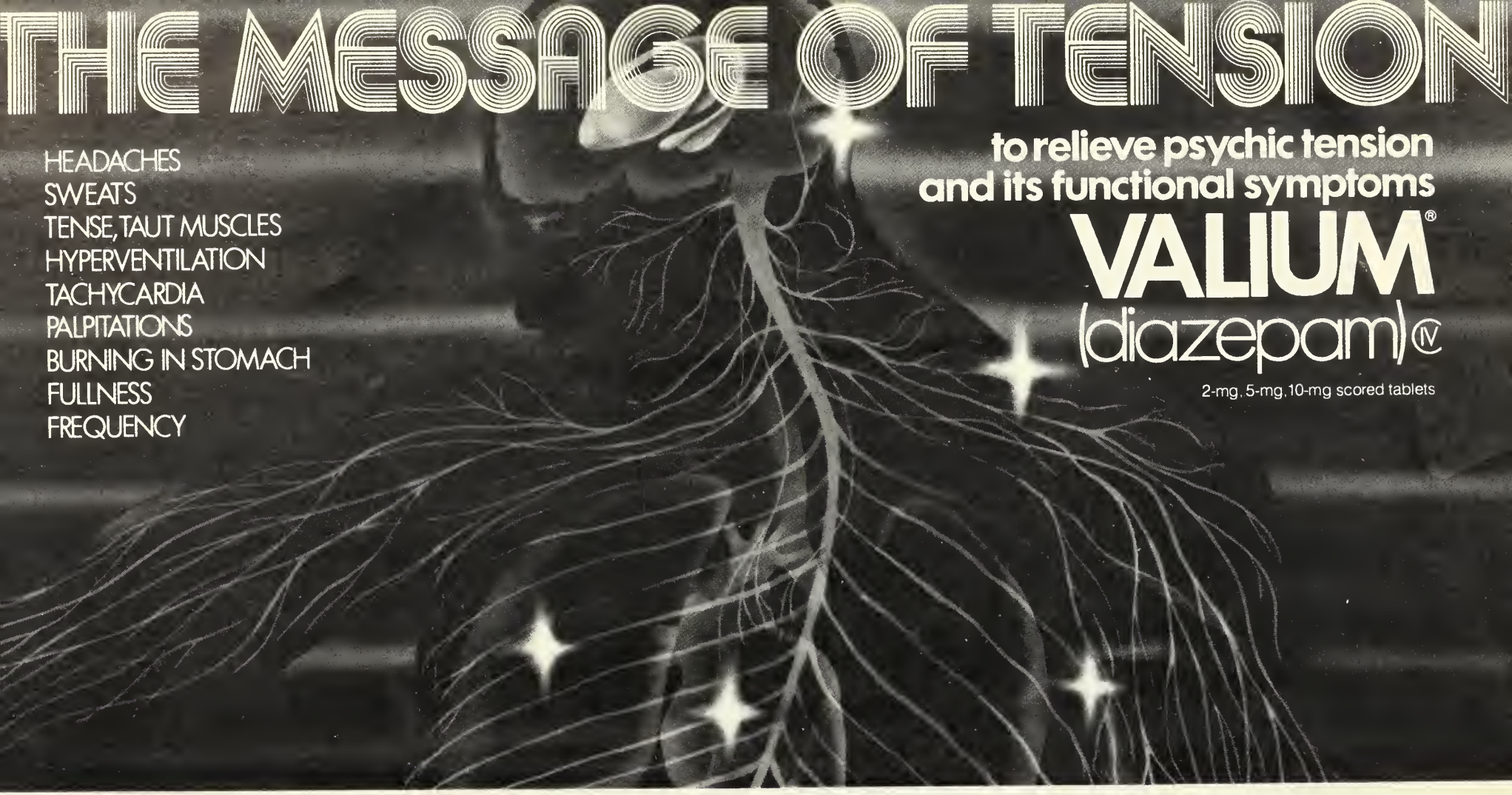
Dear KMS Member:

Several changes have been effected with this issue of the *Journal*. These changes are designed to keep us in the mainstream journalistically and to better serve you professionally.

One major change is the computerization of our membership list. The initial logistics have involved much trial-and-error education. Our efficiency in utilizing the computer is rapidly improving, but is limited by the degree of accuracy of the information we possess. Will you please help us to help you by carefully checking your individual listings (alphabetical and city) and advising us of any errors?

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VALIUM®(diazepam)

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome, convulsive disorders (not for sole therapy)
The effectiveness of Valium in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed, drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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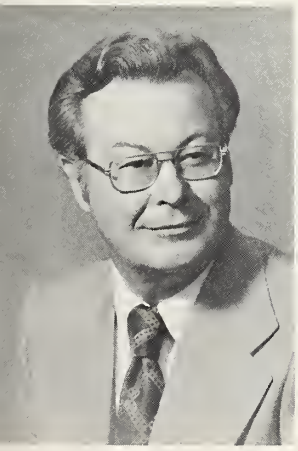
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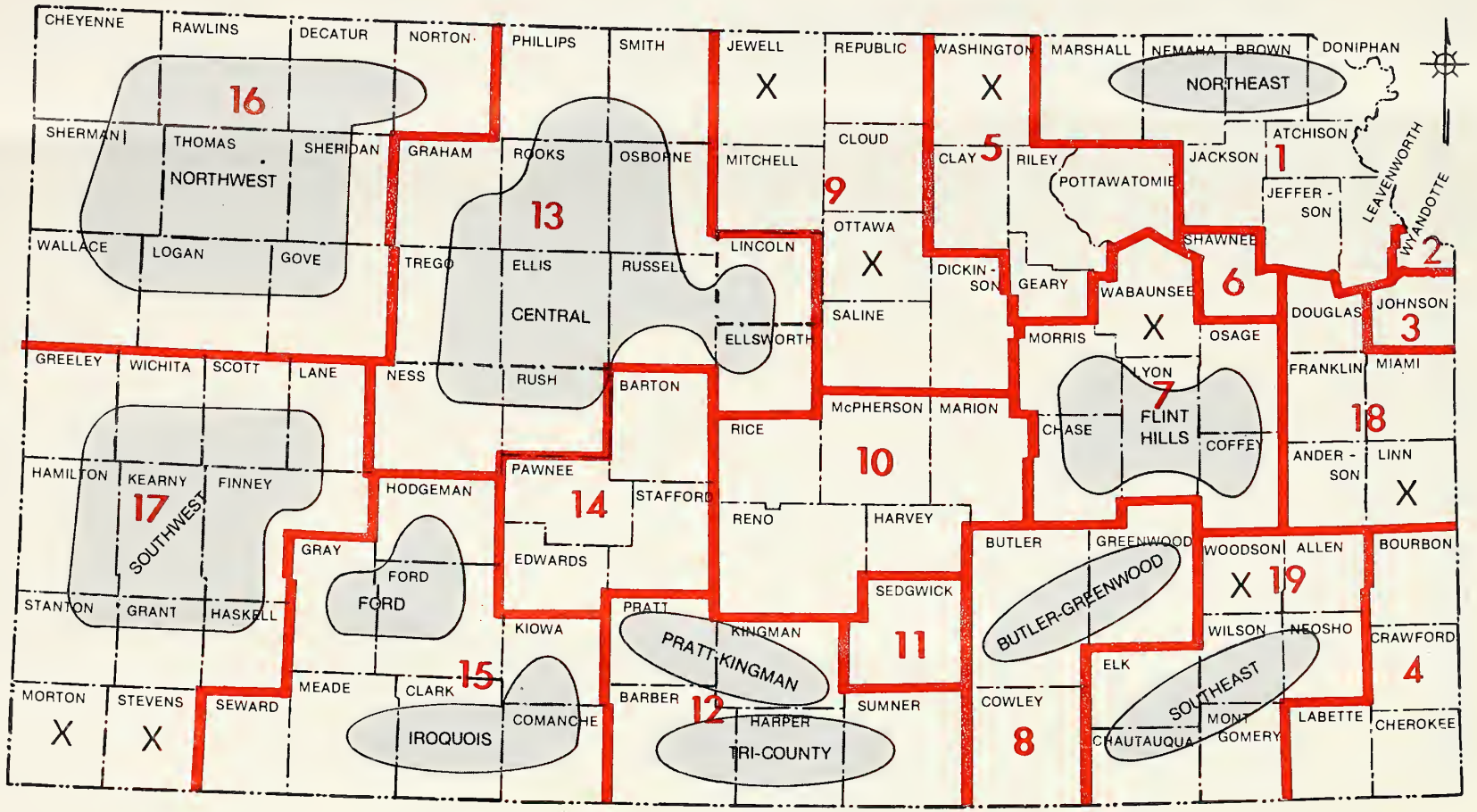


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St. Anthony Hospital — 913/625-7301
Herington — Herington Municipal Hospital — 913/258-2207
Hiawatha — Hiawatha Community Hospital — 913/742-2131
Hill City — Graham County Hospital — 913/674-2121
Hillsboro — Salem Hospital — 316/947-3114
Hoisington — Hoisington Lutheran Hospital — 316/653-2114
Holton — Holton City Hospital — 913/364-2116
Horton — Horton Community Hospital — 913/486-2142
Hoxie — Sheridan County Hospital — 913/675-3281
Hugoton — Stevens County Hospital — 316/544-8511
Hutchinson — Hutchinson Hosp. Corp. — 316/663-6811
Independence — Mercy Hospital — 316/331-2200
Iola — Allen County Hospital — 316/365-3131
Jetmore — Hodgeman County Hospital — 316/357-8361
Johnson — Stanton County Hospital — 316/492-6250
Junction City — Geary Community Hospital — 913/238-4131
Kansas City
Bethany Medical Center — 913/281-8400
Prov.-St. Margaret Health Ctr. — 913/334-2500
Univ. of Ks. Med. Ctr. — 913/588-5250
Kingman
Kingman Community Hospital — 316/532-3147
Kinsley — Edwards County Hospital — 316/659-3621
Kiowa — Kiowa District Hospital — 316/825-4130
LaCrosse — Rush County Memorial Hospital — 913/222-2545
Lakin — Kearney County Hospital — 316/355-7111
Larned
St. Joseph Memorial Hospital — 316/285-3161
Lawrence
Lawrence Memorial Hospital — 913/843-3680
Watkins Memorial Hospital — 913/843-4455
Leavenworth
Cushing Memorial Hospital — 913/682-8000
St. John's Hospital — 913/682-3721

Leoti — Wichita County Hospital — 316/375-2233
Liberal — Southwest Medical Center — 316/624-1651
Lincoln — Lincoln County Hospital — 913/524-4403
Lindsborg — Lindsborg Community Hospital — 913/227-3308
Lyons — Hospital District #1, Rice County — 316/257-2348
Manhattan
Lafene Student Health Ctr., KSU — 913/532-6544
Memorial Hospital — 913/776-3300
St. Mary Hospital — 913/776-3322
Mankato — Jewell County Hospital — 913/378-3137
Marion — St. Luke Hospital — 316/382-2177
Marysville — Community Memorial Hospital — 913/562-2311
McPherson — Memorial Hospital — 316/241-2250
Meade — Meade District Hospital — 316/873-2141
Medicine Lodge — Medicine Lodge Memorial Hospital — 316/856-3771
Minneapolis — Ottawa County Hospital — 913/392-2122
Minneola — Minneola District Hospital — 316/885-4264
Moundridge — Mercy Hospital — 316/345-6391
Neodesha — Wilson County Hospital — 316/325-2611
Ness City — Ness County District #2 — 913/798-2291
Newton
Axtell Christian Hospital — 316/283-5200
Bethel Deaconess — 316/283-2700
Prairie View Mental Health Center — 316/283-2400
Norton
Norton County Hospital — 913/877-3351
Valley Hope Alcoholic Ctr. — 913/927-5101
Oakley — Logan County Hospital — 913/672-3211
Oberlin — Decatur County Hospital — 913/475-2201
Olathe
Mid-Continent Psychiatric — 913/782-4282
Olathe-Community Hospital — 913/782-1451
Onaga — Community Hospital — 913/889-4274
Osborne — Osborne County Memorial Hospital — 913/346-2121
Oswego — Oswego Hospital — 316/795-4423
Ottawa — Ransom Memorial — 913/242-3344
Paola — Miami County Hospital — 913/294-2327
Parsons
Labette Co. Medical Center — 316/421-4880
MKT Railroad Employees' Hosp. — 316/421-2700
Phillipsburg — Phillips County Hospital — 913/543-5226
Pittsburg — Mt. Carmel Medical Center — 316/231-6100
Plainville — Plainville Rural Hospital — 913/434-4553
Pratt — Pratt County Hospital — 316/672-6476
Quinter — Gove County Hospital — 913/754-3341
Ransom — Ness County Dist. #1 — 913/731-2231
Russell — Russell City Hospital — 913/483-3131
Sabetha — Sabetha Community Hospital — 913/284-2121
Saint Francis — Cheyenne County Hospital — 913/332-2104
Saint John — St. John District Hospital — 316/549-3255
Salina
Asbury Hospital — 913/827-4411
St. John's Hospital — 913/827-5591
Satanta — Satanta District Hospital — 316/649-2200
Scott City — Scott County Hospital — 316/872-2121
Sedan — Sedan City Hospital — 316/725-3115
Seneca — Nemaha Valley Community Hospital — 913/336-2181
Shawnee Mission — Shawnee Mission Med. Ctr. — 913/676-2000
Smith Center — Smith County Memorial Hospital — 913/282-6661
Spearville — Spearville District Hospital — 316/385-2661
Stafford — Stafford District Hospital — 316/234-5221
Syracuse — Hamilton County Hospital — 316/384-7461
Topeka
Memorial Hospital — 913/354-5100
C. F. Menninger Memorial Hospital — 913/234-9566
St. Francis Hospital and Med. Ctr. — 913/295-8000
Stormont-Vail Hospital — 913/354-6111
Tribune — Greeley County Hospital — 316/376-4222
Ulysses — Bob Wilson Memorial Hospital — 316/356-1266
WaKeeney — Trego County-Lemke Memorial Hospital — 913/743-2134

Wamego — Wamego City Hosp. — 913/456-2295
Washington — Washington County Hospital — 913/325-2211
Wellington
St. Luke's Hospital — 316/326-7451
Wellington Hosp. & Clinic — 316/326-3353
Westmoreland — Dechairo Hospital — 913/457-3311
Wichita
E. B. Allen Memorial — 316/268-8211
Osteopathic Hospital — 316/943-9353
St. Francis Hospital — 316/262-6211
St. Joseph Medical Center — 316/685-1111
Wesley Medical Center — 316/685-2151
Winchester — Jefferson County Memorial Hospital — 913/774-4340
Winfield — William Newton Memorial Hospital — 316/221-2300

STATE INSTITUTIONS

Larned — Larned State Hospital — 316/285-2131
Norton — Norton State Hospital — 913/927-3301
Osawatomie — Osawatomie State Hospital — 913/755-3151
Parsons — Parsons State Hospital & Training Center — 316/421-6550
Topeka
Kansas Neurological Institute — 913/296-5301
Topeka State Hospital — 913/296-4596
Winfield — Winfield State Hospital & Training Center — 316/221-1200

VETERANS AND MILITARY HOSPITALS

Ft. Leavenworth — Munson Army Hospital — 913/684-3241
Ft. Riley — Irwin U. S. Army Hospital — 913/239-3663
Leavenworth — Veteran's Adm. Ctr. — 913/682-2000
Topeka
Veterans Administration Hospital — 913/272-3111
Wichita
Veterans Administration Hospital — 316/685-2221

POISON CONTROL CENTERS

Atchison — Atchison Hospital — 913/367-2131
Dodge City — Trinity Hospital — 316/483-8133
Emporia — Newman Memorial Hospital — 316/342-7120
Fort Scott — Mercy Hospital — Day: 316/223-3100; Night: 316/223-2200
Great Bend — Central Kansas Medical Center — Day: 316/793-3523; Night: 316/792-2511
Hays — Hadley Memorial Hospital — Day: 913/625-2515, Ext. 237; Night: 913/625-3441
Kansas City
University of Kansas Medical Center — 913/588-6633
Bethany Hospital — 913/621-6600
Lawrence — Lawrence Memorial Hospital — Day: 913/843-3680 or 842-4477; Night: 913/843-5874
Parsons — Labette County Medical Center — 316/421-4880
Salina — St. John's Hospital — 913/827-5591, Ext. 222
Topeka — Stormont-Vail Hospital — 913/354-6100
Wichita — Wesley Hospital — 316/685-2151, Ext. 377
Kansas Poison Control Information Center:
Kansas Department of Health, Food and Drug Division, Topeka — Evan Wright, Director — 913/862-9360, Ext. 541
Antivenin Index Center — 405/271-5454

HOME HEALTH AGENCIES

Anderson County Hospital, Garnett 60032 — 913/448-3131
Barton County Health Dept., Courthouse, Great Bend 67530 — 316/793-7879
Butler-Greenwood County Health Dept., Courthouse, El Dorado 67042 — 316/321-3400
Clay County Hospital, Clay Center 67432 — 913/632-2144
Cloud County Health Dept., Courthouse, Concordia 66901 — 913/243-3588
Coffey County Health Dept., Courthouse, Burlington 66839 — 316/364-5831
Douglas County Visiting Nurses' Association, 342 Missouri St., Lawrence 66044 — 913/843-3738
Ellis County-St. Anthony Hospital, Hays 67601 — 913/625-2556
Ellsworth County Health Dept., Courthouse, Ellsworth 67439 — 913/472-4234
Ford County Health Dept., Courthouse, Dodge City 67801 — 316/225-4991
Franklin County Health Dept., 112 W. Tecumseh, Ottawa 66067 — 913/242-1873
Graham County Hospital, Hill City 67642 — 913/674-2121
Harper County Health Dept., Courthouse, Anthony 67003 — 316/842-5264
Harvey County Health Dept., Courthouse, Newton 67114 — 316/283-1060

Jackson County Health Dept., Courthouse, Holton 66436 — 913/364-2670

Jefferson County Health Dept., Courthouse, Oskaloosa 66066 — 913/863-2447

Kansas City Visiting Nurses' Association, One Gateway Center, Suite 219, Kansas City, Kansas 66117 — 913/371-3770

Kingman County Health Dept., Courthouse, Kingman, 67068 — 316/532-2221

Labette County Health Dept., Box 786, Parsons 67357 — 316/421-4350

Logan County Hospital, Oakley 67748 — 913/672-3211

Lyon County Home Health Service, Newman Memorial Hospital, Emporia 66801 — 316/342-8562

McPherson County Health Dept., Courthouse, McPherson 67460 — 316/241-1753

Morris County Health Dept., Courthouse, Council Grove 66846 — 316/767-5175

Osage County Health Dept., Courthouse, Lyndon 66451 — 913/828-3281

Pratt County Health Dept., 106 E. 2nd, Pratt 67124 — 316/672-6122

Reno County-St. Elizabeth Hospital, 500 W. 20th, Hutchinson 67501 — 316/665-5531

Republic County Health Dept., Courthouse, Belleville 66935 — 913/527-5385

Rice County Health Dept., Courthouse, Lyons 67554 — 316/257-2171

Riley County Health Dept., 616 Poyntz, Manhattan 66502 — 913/776-9721

Russell County Health Dept., Courthouse, Russell 67665 — 913/483-2641

Salina-Saline County Health Dept., 300 W. Ash, Salina 67401 — 913/827-9376

Sedgwick County Health Dept., 1900 E. 9th, Wichita 67214 — 316/268-8433

Shawnee County Health Dept., 1615 W. 8th, Topeka 66606 — 913/233-8961

Sheridan County Hospital, Hoxie 67740 — 913/675-3281

Thomas County-St. Thomas Hospital, Colby 67701 — 913/462-3335

Washington County Health Dept., Washington 66968 — 913/325-2600

GENETIC COUNSELING CENTERS

Kansas City — Genetic Counseling Center, Division of Medical Genetics, K.U.M.C., 39th & Rainbow Blvd., Kansas City, KS 66103 — 913/588-6043 — R. Neil Schimke, M.D., Director

Topeka — Genetic Counseling Center, 105-A Medical Arts Bldg., 1001 Horne, Topeka, KS 66604 — 913/232-1295 — David E. Gray, M.D., Director

Wichita — Genetic Counseling Unit, Wesley Medical Center, 550 N. Hillside, Wichita, KS 67214 — 316/685-2151

How to Ruin an Organization

1. Stay away from all meetings.
2. If you do come, find fault.
3. Decline office or appointment to a committee.
4. Get sore if you aren't nominated or appointed.
5. After you are named, don't attend board or committee meetings.
6. If you get to one, despite your better judgment, clam up until it's over. Then sound off on how things really should be done.
7. Do not work if you can help it. When the Old Reliables pitch in, accuse them of being a clique.
8. Oppose all banquets, parties, and shindigs as being a waste of members' money.
9. If everything is strictly business, complain that the meetings are dull and the officers a bunch of old sticks.
10. Never accept a place at the head table.
11. If you aren't asked to sit there, threaten to resign because you aren't appreciated.
12. Don't rush to pay your dues. Let the directors sweat; after all, they wrote the budget.
13. Don't preregister for meetings. Say "I thought sure you'd know I was coming." Then complain loudly that registration procedures at "too slow."

NOTE: The source of the above is unknown, but we would like to add a 14th step:

14. Never send articles to your *Journal* and editors, and then complain that Society publications do not contain enough input from members.



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I swear by Apollo the physician, and Aesculapius, and Health, and All-heal, and all the gods and goddesses, that, according to my ability and judgment, I will keep this Oath and this stipulation—to reckon him who taught me this Art equally dear to me as my parents, to share my substance with him, and relieve his necessities if required; to look upon his offspring in the same footing as my own brothers, and to teach them this art, if they shall wish to learn it, without fee or stipulation; and that by precept, lecture, and every other mode of instruction, I will impart a knowledge of the Art to my own sons, and those of my teachers, and to disciples bound by a stipulation and oath according to the law of medicine, but to none others. I will follow that system of regimen which, according to my ability and judgment, I consider for the benefit of my patients, and abstain from whatever is deleterious and mischievous. I will give no deadly medicine to any one if asked, nor suggest any such counsel; and in like manner I will not give to a woman a pessary to produce abortion. With purity and with holiness I will pass my life and practice my Art. I will not cut persons laboring under the stone, but will leave this to be done by men who are practitioners of this work. Into whatever houses I enter, I will go into them for the benefit of the sick, and will abstain from every voluntary act of mischief and corruption; and, further, from the seduction of females or males, of freemen and slaves. Whatever, in connection with my professional practice or not, in connection with it, I see or hear, in the life of men, which ought not to be spoken of abroad, I will not divulge, as reckoning that all such should be kept secret. While I continue to keep this Oath unviolated, may it be granted to me to enjoy life and the practice of the art, respected by all men, in all times! But should I trespass and violate this Oath, may the reverse be my lot!



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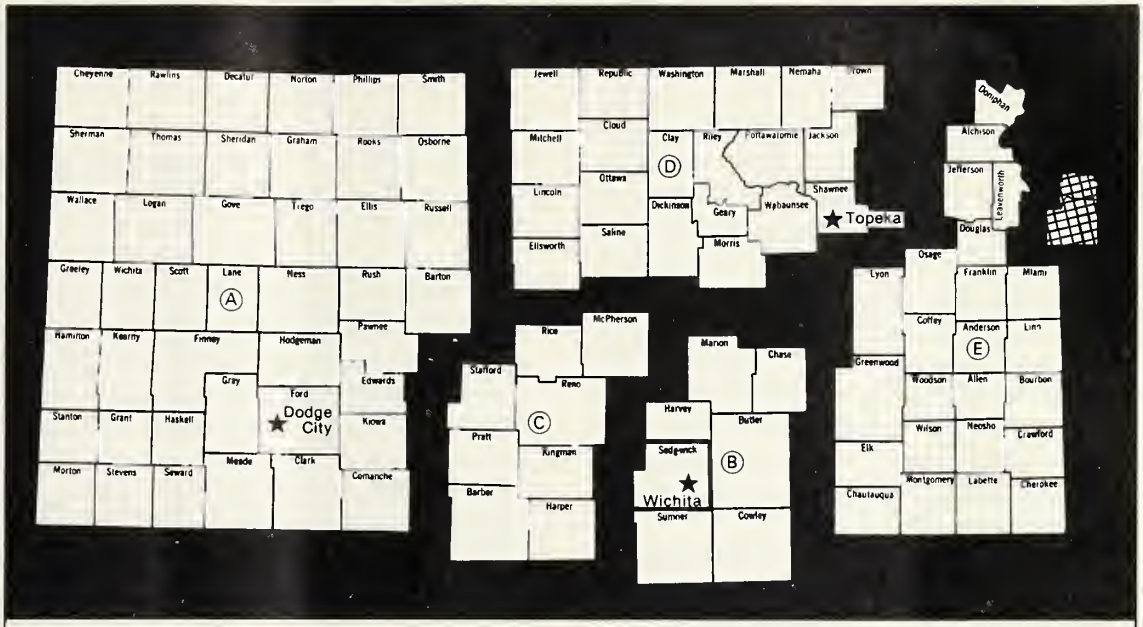
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KMS Impaired Physician Program

Statement of Purpose

We believe that the problem of impaired professional performance among physicians is the responsibility of the medical profession, requiring us to deal with such physicians unequivocally, fully, and compassionately with all appropriate means at our disposal. To meet this need, we recommend that a committee be established along with a comprehensive action program.

The purpose of the Committee for the Impaired Physician Program is to:

1. Establish a statewide non-coercive program for locating, contacting, and offering rehabilitative help to physicians who have become professionally impaired to varying degrees because of alcoholism, other drug dependence, mental, physical and aging problems, and/or medical incompetence.
2. Function as a peer review organization.
3. Continue to work in liaison with the State Board of Healing Arts as the formal agency with the legal authority to deal with the impaired physician.
4. Establish programs of education and prevention concerned with alcoholism, other drug dependence, mental, physical and aging problems, and/or medical incompetence.

General Principles

Regarding the impaired physician, the Kansas Medical Society subscribes to the following guiding principles:

1. We are motivated in this area by humanitarian concern for the public and the impaired physician and his family.
2. We recognize that alcoholism, other drug dependence, mental, physical and aging problems, and/or medical incompetence represent forms of physician impairment that may be ignored or untreated.
3. We recognize that most alcoholism, other drug dependence, mental, physical and aging problems and/or medical incompetence are treatable conditions.
4. As practitioners of the healing arts, we, more than any other group, should favor treatment and/or rehabilitation of our impaired colleagues above any other alternative.
5. We encourage all impaired physicians to seek help and cooperate in treatment by all means at the disposal of the Kansas Medical Society.
6. We favor the *earliest possible* intervention in cases of physician impairment while personal, financial, mental, and physical resources are intact and minimum damage has been done to professional reputation or to the public.
7. All Kansas Medical Society action taken on behalf of the impaired physician will be done in such a manner as to preserve, unimpaired, the physician's right to continue practice without restriction or stigma upon recovery or remission.
8. All actions taken in the area of impaired physicians by the Kansas Medical Society are intended in the best interest of the physician and the public.
9. Referral to the State Board of Healing Arts for sanctions will be considered only when the impaired physician's actions endanger the public or himself and he repeatedly refuses assistance. When a referral is made to the Kansas State Board of Healing Arts by the Committee for the Impaired Physician Program, the State Board of Healing Arts will act to suspend the license of the physician in question according to the laws governing such licensure. Any temporary suspension or limitation order would take effect when served in person on the licensee.

Structure

I. The Impaired Physician Program of the Kansas Medical Society will encompass the entire State of Kansas and be administered by the Kansas Medical Society. Existing mechanisms on a local level will be integrated and utilized by the statewide program, but the main impetus for continuing the program must come from the state society.

II. There will be established two committees: the Committee for the Impaired Physician Program and the Physician Contact/Support Committee.

A. *The Committee for the Impaired Physician Program:* This Committee will consist of ten (10) physicians who are knowledgeable and vitally interested in the subject of physician impairment. Additional members may be added at the discretion of the Committee for a designated length of time.

Appointed terms of adequate length should be established to ensure continuity; additionally, the appointments should be on a rotating basis. The Committee should be established as a standing committee of the Executive Committee of The Kansas Medical Society and accountable to the executive committee.

The Committee for the Impaired Physician Program will be responsible to:

1. Broadly publicize the Impaired Physician Program and direct an educational effort to physicians and their families, hospital staffs, county medical societies, auxiliary, and others to communicate the general principles of the program and solicit cooperation;

2. Establish a registry of the best appropriate resources and therapists in the areas of alcoholism, other drug dependence, mental health, geriatrics, and/or medical incompetence. This registry will be continually updated as necessary and will serve as the basis for designating intervention teams;

3. Supply current factual information concerning these conditions and be able to make knowledgeable referrals for effective treatment.

4. Confirm complaints of physician impairment and then refer to a Contact/Support team for motivation into voluntary treatment;

5. Evaluate the quality and efficiency of services rendered by physicians referred to the Committee for the Impaired Physician Program;

6. Recommend overall policy and develop guidelines regarding the implementation and maintenance of the Impaired Physician Program;

7. Establish criteria for participating as a Contact/Support team member and be responsible for quality control of this membership;

8. Require follow-up information to be provided by the Contact/Support teams to determine the current status of the impaired physician who has begun treatment;

9. Conduct routine administrative functions including fiscal management and recommendation of needed budget;

10. Make recommendations to the State Board of Healing Arts regarding any impaired physician when a majority of the Committee agrees that the reasonable efforts of the Contact/Support teams have failed to produce an acceptable degree of rehabilitation.

B. *Physician Contact/Support Committee:* There will be established a larger group from which Contact/Support team members are drawn. The names of these people should be maintained by the Committee for the Impaired Physician Program in a resource registry. Contact/Support team members should be drawn from all areas of the state, should have expertise in the areas of alcoholism, other drug dependence, mental health, geriatrics and/or medical incompetence and some should themselves be rehabilitated impaired physicians. When the Committee for the Impaired Physician Program deems it appropriate, a Contact/Support team will be selected in the following manner: Where evidence exists that a physician's ability to function professionally is impaired, the Committee will select a Contact/Support team. At least one member of this team will be a physician from the resource registry. The registry may be the source of the entire team. No member of the team will be involved in the ongoing treatment process.

It is often advisable that physician Contact/Support members be drawn from a locale outside of the geographic area of the impaired physician's practice.

It will be the responsibility of the Contact/Support team to:

1. Meet with family members and others in appropriate settings, with the impaired physician and present him with documentation that impairment exists; and to express the concerns of his colleagues and family;

2. Encourage the impaired physician to seek help voluntarily, or assist him into active treatment;

3. Maintain contact with the impaired physician to support and encourage his cooperation in the treatment and in the case of the alcohol and/or other drug dependent physician for active participation in Alcoholics Anonymous; and encourage membership in International Doctors in Alcoholics Anonymous (IDAA), and the American Medical Society on Alcoholism;

4. Make every effort to assist the impaired physician to continue his professional duties insofar as he is considered able to do so by the physician in charge of treatment;

5. Attempt to plan for, or to help, provide for opportunities for the impaired physician to continue limited duties, supervised if necessary, as thought reasonable by his treating physician;

6. In cases where the impaired physician has discontinued or limited his work, aid him to return to appropriate work duties as soon as possible;

7. Where there is a failure to seek treatment and/or failure to cooperate after a reasonable period of time, this fact will be reported to the Chairman of the Committee for the Impaired Physician Program.

III. Referral calls concerning any alleged impaired physician shall be made to the Kansas Medical Society Headquarters in Topeka. Call collect

(913) 235-2383. This number will be answered 24 hours a day.

A person reporting an alleged impaired physician will be asked: (1) the name of the alleged impaired physician; and (2) the general nature of the complaint. The caller will be asked to identify himself if he wishes, but will be assured that his complaint is confidential and that his name will not be revealed to the alleged impaired physician without the caller's knowledge and consent.

Following receipt of any call, the Kansas Medical Society office staff will contact the Chairman of the Committee for the Impaired Physician Program, or his designee, and report the basic information obtained from the complainant. The Chairman or his designee will telephone an appropriate member of the county medical society in the alleged impaired physician's Council district to begin the process of verification and/or assessment of the need to pursue the complaint with a Contact/Support team.

A. *Voluntary or Self-referral:* When the impaired physician contacts the Kansas Medical Society program directly, the following steps will take place:

1. The appropriate staff member receiving the call contacts the Chairman or his designee of the Committee for the Impaired Physician Program;

2. The Chairman or his designee arranges for a member of this Committee to contact the impaired physician to inquire about the nature of his illness or problem, and to discuss with the physician his willingness to undertake appropriate treatment;

3. The Chairman or his designee then assigns a Contact/Support team who will become advocate for the physician as he undergoes treatment. The steps outlined under the "responsibilities of the Contact/Support team," (points 3-7), will then go into effect.

B. *Involuntary or Other Means of Referral:* The Kansas Medical Society will accept from any responsible source (physician peer, hospital administrator, patient, physician's spouse, etc.) information indicating that a physician may be impaired. *Specific details regarding the physician involved will not be sought or accepted at the time of the original call.* In such a case the following steps will take place:

1. The appropriate staff member receiving the call contacts the Chairman of the Committee for the Impaired Physician Program, or his designee; and reports: (1) the name of the alleged impaired physician; (2) the general nature of the complaint; and (3) the caller's name and telephone number if volunteered by the caller.

2. The chairman or his designee contacts an appropriate member(s) of the county medical society in the alleged impaired physician's council district to inquire about the alleged impaired physician along with the specific reasons for the alleged impairment.

3. The Committee member then evaluates, with the assistance of other members of the Committee as he deems necessary, the information, making as much use as possible of firsthand information concerning the specific incidents of the demonstrated impairment. All such information gathered by the Committee for the Impaired Physician Program will be kept confidential.

4. Having determined sufficient cause exists to justify contacting the physician thought to be impaired, the Committee by direction of its Chairman or his designee will select a Contact/Support team. Steps 1-7 under "responsibilities of the Contact/Support team," then go into effect.

C. *Conclusion of Intervention.* When a physician referred to the Kansas Medical Society is considered rehabilitated, and restored to medical practice, the Kansas Medical Society intervention is concluded. This may require a period of anywhere from 2 to 3 or more years. All records are then destroyed.

When all efforts fail and the degree of impairment of the physician's professional performance threatens the public trust, the matter will be reported to the Kansas State Board of Healing Arts by the Chairman of the Committee for the Impaired Physician Program.

Legal Issues

1. *Immunity from Liability:* Exemption from liability for all those who are actively involved in the program, including those who in good faith report cases of physician impairment, the Kansas Medical Society committees for the Impaired Physician Program and the Contact/Support Teams, the Kansas Medical Society itself, both generally and including the Executive Committee of The Kansas Medical Society, as well as any of the committees, staff, counsels, or commissions of this Society otherwise involved in the program, is provided through the following mechanisms:

KSA 65-2898 and KSA 65-4909 provide immunity by relieving any person reporting to the State Board of Healing Arts and other licensing boards from civil liability for reporting any alleged incidents of malpractice or the qualifications, fitness or character of persons licensed or registered by such boards, if such report is made under oath and in good faith.

The bills also provide that any state, regional, or local association composed of persons licensed to practice the healing arts, or other health care providers, or any individual members or committees

thereof which investigate and communicate information pertaining to alleged malpractice incidents or the qualifications, fitness or character of any licensee or registrant to the appropriate licensing board, shall be immune from liability in civil actions if the investigation and communication were made in good faith and did not represent as true any matter not reasonably believed to be true. On this basis, members of the Committee for the Impaired Physician Program and the physician Contact/Support Committee, as well as others reporting, are granted immunity, so long as the conditions of the statute are met.

2. *Confidentiality:* The purpose of the Kansas Medical Society program for impaired physicians is to offer early intervention and help for impaired physicians, at the same time performing a peer review function by evaluating the quality and efficiency of services rendered by physicians referred to the Committee for the Impaired Physician Program. The entire program should be carried out with minimal disruption of the physician's practice, and no harm to his reputation. For these reasons, all information gathered on the physician in question must be held in the strictest confidence and any investigation of reports carried out discreetly. Proper records must be maintained but only in strictest confidence and protection.

3. *Relationship to the State Board of Healing Arts:* The Kansas Legislature enacted HB-2008 in 1975, which grants the Healing Arts Board the authority to utilize the AMA Model Act on the impaired physician. This act was drafted by AMA in 1974, and allows each individual state board considerable latitude and authority to act in the area of physician impairment. It is clear that the trend seems to be in the direction of closer cooperation between state medical associations and licensing bodies along the entire continuum of detection, diagnosis, treatment and rehabilitation.

HB-3109, enacted in 1978, grants the Healing Arts Board authority for temporary suspension and limitation of license of any licensee, without notice or hearing, if the Board determines that there is cause to believe that grounds exist for such revocation, suspension or limitation, and that the licensee's continuation in practice would constitute an imminent danger to the public health and safety. Any temporary suspension or limitation order would take effect when served in person on the licensee.

The Board will institute proceedings for a hearing simultaneously with the Board's action to limit or suspend the license temporarily. Such hearing is to be held within fifteen (15) days from the date of the temporary limitation or suspension unless a continuance is requested by the licensee.

In no case shall a temporary suspension or temporary limitation of a license be in effect for a period of time in excess of 90 days. At the end of such period of time, the licensee shall be reinstated to full licensure unless the Board has revoked, suspended or limited the license.

With the support and cooperation of the State Board of Healing Arts, the Kansas Medical Society's program includes non-member physicians. The alternative for the impaired non-member would be either access to the Kansas Medical Society's mechanism or in the event of failure to accept this help, referral to the State Board's formal mechanism. With the State Board of Healing Arts in the background as the "court of last resort," the Kansas Medical Society's efforts should be more effective in the great majority of instances of physician impairment.

Frank Bichlmeier, M.D., Co-Chairman	Kansas City
George M. Penn, M.D., Co-Chairman	Topeka
John Cody, M.D.	Hays
H. Ivor Jones, M.D.	Shawnee Mission
Roy Neil, M.D.	Hays
Ivan E. Rhodes, M.D.	Wichita
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Max E. Teare, M.D.	Garden City
Kermit G. Wedel, M.D.	Minneapolis
Jackie Burnett, R.N., Auxiliary Liaison	Halstead
Mary Ann Penn, R.N., Auxiliary Liaison	Topeka

Principles of Medical Ethics of the American Medical Association

PREAMBLE

These principles are intended to aid physicians individually and collectively in maintaining a high level of ethical conduct. They are not laws but standards by which a physician may determine the propriety of his conduct in his relationship with patients, with colleagues, with members of allied professions, and with the public.

Section 1

The principal objective of the medical profession is to render service to humanity with full respect for the dignity of man. Physicians should merit the confidence of patients entrusted to their care, rendering to each a full measure of service and devotion.

Section 2

Physicians should strive continually to improve medical knowledge and skill, and should make available to their patients and colleagues the benefits of their professional attainments.

Section 3

A physician should practice a method of healing founded on a scientific basis; and he should not voluntarily associate professionally with anyone who violates this principle.

Section 4

The medical profession should safeguard the public and itself against physicians deficient in moral character or professional competence. Physicians should observe all laws, uphold the dignity and honor of the profession and accept its self-imposed disciplines. They should expose, without hesitation, illegal or unethical conduct of fellow members of the profession.

Section 5

A physician may choose whom he will serve. In an emergency, however, he should render service to the best of his ability. Having undertaken the care of a patient, he may not neglect him; and unless he has been discharged he may discontinue his services only after giving adequate notice. He should not solicit patients.

Section 6

A physician should not dispose of his services under terms or conditions which tend to interfere with or impair the free and complete exercise of his medical judgment and skill or tend to cause a deterioration of the quality of medical care.

Section 7

In the practice of medicine a physician should limit the source of his professional income to medical services actually rendered by him, or under his supervision, to his patients. His fee should be commensurate with the services rendered and the patient's ability to pay. He should neither pay nor receive a commission for referral of patients. Drugs, remedies or appliances may be dispensed or supplied by the physician provided it is in the best interests of the patient.

Section 8

A physician should seek consultation upon request; in doubtful or difficult cases; or whenever it appears that the quality of medical service may be enhanced thereby.

Section 9

A physician may not reveal the confidences entrusted to him in the course of medical attendance, or the deficiencies he may observe in the character of patients, unless he is required to do so by law or unless it becomes necessary in order to protect the welfare of the individual or of the community.

Section 10

The honored ideals of the medical profession imply that the responsibilities of the physician extend not only to the individual, but also to society where these responsibilities deserve his interest and participation in activities which have the purpose of improving both the health and the well-being of the individual and the community.

Legal Definition of Death

K.S.A. 77-202. Definition of Death.

A person will be considered medically and legally dead if, in the opinion of a physician, based on ordinary standards of medical practice, there is the absence of spontaneous respiratory and cardiac function and, because of the disease or condition which caused, directly or indirectly; these functions to cease, or because of the passage of time since these functions ceased, attempts at resuscitation are considered hopeless; and, in this event, death will have occurred at the time these functions ceased; or

A person will be considered medically and legally dead if, in the opinion of a physician, based on ordinary standards of medical practice, there is the absence of spontaneous brain function; and if based on ordinary standards of medical practice, during reasonable attempts to either maintain or restore spontaneous circulatory or respiratory function in the absence of aforesaid brain function, it appears that further attempts at resuscitation or supportive maintenance will not succeed, death will have occurred at the time when these conditions first coincide. Death is to be pronounced before artificial means of supporting respiratory and circulatory function are terminated and before any vital organ is removed for purposes of transplantation.

These alternative definitions of death are to be utilized for all purposes in this state, including the trials of civil and criminal cases, any laws to the contrary notwithstanding.

To Contact Your Legislators:

U. S. CONGRESSIONAL DELEGATION

Senators:

James B. Pearson, 5313 Dirksen Senate Office Bldg., 20515. Phone extension: 4774

Robert Dole, 4213 Dirksen Senate Office Bldg., 20515. Phone extension: 6521

Representatives:

1. Keith G. Sebelius, 1211 Longworth House Office Bldg., 20510. Phone extension: 2715
2. Martha Keys, 1207 Longworth House Office Bldg., 20510. Phone extension: 6601
3. Larry Winn, Jr., 2430 Rayburn House Office Bldg., 20515. Phone extension: 2865
4. Daniel R. Glickman, 1128 Longworth Bldg., 20515. Phone extension: 2216
5. Joe Skubitz, 2211 Rayburn House Office Bldg., 20515. Phone extension: 3911

When writing, the following form is appropriate:

Senators:

Honorable John Doe,
United States Senate
Address

Dear Senator Doe:

Representatives:

Honorable John Doe,
House of Representatives
Address

Dear Mr. Doe:

To telephone Senators and Representatives in Washington, dial or call Area Code 202, 224 plus the four-digit extension shown above with the Senator's name. To reach a Representative, dial 225, plus the extension number.

KANSAS LEGISLATURE

To write state Senators and Representatives, the following addresses may be used:

Senators:

Honorable John Doe
Senate Chambers
State Capitol Building
Topeka, KS 66612

Dear Senator Doe:

Phone: (913) 296-7300

Representatives:

Honorable John Doe
House of Representatives
State Capitol Building
Topeka, KS 66612

Dear Representative Doe:

Phone: (913) 296-7500

Good Samaritan Act

K.S.A. 65-2891. Emergency care or assistance at scene of accident by certain persons, including treatment of minors; liability in damages; emergency care or assistance to minors engaging in sports without parental consent; damage liability; temporary emergency care or assistance during emergency within a hospital; liability; ordinary standards of care and rules of negligence applicable to emergency care and treatment.

(a) Any physician or any other practitioner of the healing arts, or dentist licensed to practice under the laws of this state, or any other state, or any registered professional nurse, or any physicians' assistant who has successfully completed an American medical association approved training program and has successfully completed the national board examination for physicians' assistants of the American board of medical examiners or any person who has successfully completed an approved emergency service program as defined by K.S.A. 1975 Supp. 65-2891a, who in good faith renders emergency care or assistance at the scene of an emergency or accident including treatment of a minor without first obtaining the consent of the parent or guardian of such minor shall not be liable for any civil damages for acts or omissions other than damages occasioned by gross negligence or by willful or wanton acts or omissions by such person in rendering such emergency care.

(b) Any physician, registered professional nurse or dentist licensed under the laws of this state, or of any other state, to practice medicine, professional nursing, dentistry or surgery, or any physicians' assistant who has successfully completed an American medical association approved training program and has successfully completed the national board examination for physicians' assistants of the American board of medical examiners, may, in good faith render emergency care or assistance, without compensation, to any minor requiring such care or assistance as a result of having engaged in competitive sports, without first obtaining the consent of the parent or guardian of such minor. Such physician, registered professional nurse, dentist or physicians' assistant shall not be liable for any civil damages other than damages occasioned by gross negligence or by willful or wanton acts or omissions by such person in rendering such emergency care.

(c) Any physician, registered professional nurse or dentist licensed under the laws of this state or any other state to practice medicine, professional nursing, dentistry or surgery, or any physicians' assistant who has successfully completed an American medical association approved training program and has successfully completed the national board examination for physicians' assistants of the American board of medical examiners, may in good faith render emergency care or assistance during an emergency which occurs within a hospital or elsewhere, with or without compensation, until such time as the physician employed by the patient or by his or her family or by his or her guardian assumes responsibility for such patient's professional care. The physician, registered professional nurse, dentist or physicians' assistant rendering such emergency care shall not be held liable for any civil damages other than damages occasioned by negligence.

(d) Any provision herein contained notwithstanding, the ordinary standards of care and rules of negligence shall apply in those cases wherein emergency care and assistance is rendered in any physician's or dentist's office, clinic, emergency room or hospital with or without compensation. [K.S.A. 65-2891; L. 1973, ch. 252, §1; L. 1975, ch. 326, §1; July 1.]

K.S.A. 65-2891a. Same; emergency service program defined. As used in this act, emergency service program means a program of instruction, approved by the university of Kansas medical center, consisting of eighty (80) clock hours or the equivalent thereof, of preliminary emergency medical care and at least eight (8) clock hours annually of supplemental instruction.

Performance of Autopsy

65-2893. Autopsies; performance of; authorization. In any case of death wherein notification of the coroner is not required by K.S.A. 19-1031 and amendments thereto, or any case in which the coroner does not elect to perform an autopsy, an autopsy may be performed upon the body of a deceased person by a physician or surgeon when so authorized, in writing by the decedent during his lifetime. Additionally, unless the physician or surgeon has knowledge that contrary directions have been given by the decedent, the following persons in the order of priority stated, may consent to the performance of an autopsy: (1) The spouse, if one survives and if not incapacitated. If no spouse survives or if the spouse is incapacitated;

(2) an adult child;

(3) either parent;

(4) an adult brother or sister;

(5) the guardian of the decedent at the time of his death;

(6) any other person or agency authorized or under obligation to dispose of the body.

If there is no surviving spouse and an adult child is not immediately available at the time of death, the autopsy may be authorized by either parent; if a parent is not immediately available, it may be authorized by any adult brother or sister: *Provided,* That such autopsy shall not be performed under a consent given as required by a member of the class listed in (2), (3) or (4) above, if, before such autopsy is performed, any member of the class shall object to the performance of such autopsy in writing to the physician or surgeon by whom the autopsy is to be performed.

Treatment of Minors

65-2892. Examination and treatment of persons under 18 for venereal disease; liability. Any physician, upon consultation by any person under eighteen (18) years of age as a patient, may, with the consent of such person who is hereby granted the right of giving such consent, make a diagnostic examination for venereal disease and prescribe for and treat such person for venereal disease including prophylactic treatment for exposure to venereal disease whenever such person is suspected of having a venereal disease or contact with anyone having a venereal disease. All such examinations and treatment may be performed without the consent of, or notification to, the parent, parents, guardian or any other person having custody of such person. Any physician examining or treating such person for venereal disease may, but shall not be obligated to, in accord with his opinion of what will be most beneficial for such person, inform the spouse, parent, custodian, guardian or fiancé of such person as to the treatment given or needed without the consent of such person. Such informing shall not constitute libel or slander or a violation of the right of privacy or privilege or otherwise subject the physician to any liability whatsoever. In any such case, the physician shall incur no civil of criminal liability by reason of having made such diagnostic examination or rendered such treatment, but such immunity shall not apply to any negligent acts or omissions. The physician shall incur no civil or criminal liability by reason of any adverse reaction to medication administered, provided reasonable care has been taken to elicit from such person under eighteen (18) years of age any history of sensitivity or previous adverse reaction to the medication.

65-2892a. Examination and treatment of minors for drug abuse, misuse or addiction; liability. Any physician licensed to practice the healing arts in Kansas, upon consultation with any minor as a patient, may examine and treat such minor for drug abuse, misuse or addiction if such physician has secured the prior consent of such minor to the examination and treatment. All such examinations and treatment may be performed without the consent of any parent, guardian or other person having custody of such minor, and all minors are hereby granted the right to give consent to such examination and treatment. In any such case, the physician shall incur no civil or criminal liability by reason of having made such diagnostic examination or rendered such treatment, but such immunity shall not apply to any negligent acts or omissions. The physician shall incur no civil or criminal liability by reason of any adverse reaction to medication administered, if reasonable care has been taken.

Continuing Medical Education

The 1976 Kansas Legislature enacted a law requiring continuing medical education as a requisite to relicensure in Kansas. HB-2723 provides that, "From and after July 1, 1978, the Kansas State Board of Healing Arts shall require every licensee to submit with the renewal application evidence of satisfactory completion of a program of continuing education required by the Board." Physicians not in the active practice of the healing arts in the State of Kansas may be exempted from the continuing medical education requirements for relicensure by signing an affidavit furnished by the Kansas State Board of Healing Arts. The following is a description of the CME requirements established by the Board, and an explanation of how to comply with the requirements.

As a basic requirement, the Board adopted the CME requirements of the Kansas Medical Society, which is the attainment of the American Medical Association "Physician's Recognition Award." The basic criteria for the PRA is 150 hours of educational experience over a three-year period. The educational activities are described in six separate categories. Below is a brief description of each category and the maximum number of hours that can be obtained in each category:

	Credit Hours Limit
Category 1 — CME activities with accredited sponsorship (60 hours required)	no limit
Category 2 — CME activities with non-accredited sponsorship	45 hrs.
Category 3 — Medical teaching	45 hrs.
Category 4 — Papers, publications, books, exhibits	40 hrs.
Category 5 — Non-supervised individual CME activities	45 hrs.
Category 6 — Other meritorious learning experiences	45 hrs.

Completion of AMA-PRA satisfies the requirement established by the Board. Because relicensure is on a yearly basis, and the PRA is on a three-year basis, a physician need only have a valid PRA in effect to be in compliance with the requirements of the Board.

Additionally, completion of the CME certification programs of any of the following organizations also constitutes compliance with the requirements of the Board:

American Academy of Dermatology
American Association of Neurological Surgeons
American College of Emergency Physicians
American College of Radiology
American Society of Colon and Rectal Surgeons
American Academy of Family Physicians
American College of Obstetricians and Gynecologists
College of American Pathologists/American Society of Clinical Pathologists
American Psychiatric Association
Any other specialty organization CME certification programs subsequently recognized by the AMA.

If you satisfy the requirements of any of the above organizations, you need *not* apply for the "Physician's Recognition Award." Each specialty organization will notify the Kansas Medical Society of your certification, and the Kansas Medical Society will in turn notify the Board that you have satisfactorily completed your requirements.

If you do not qualify for the specialty organization programs listed above, you must apply directly to the AMA for the "Physician's Recognition Award." Upon receipt of the award, the AMA will notify the Kansas Medical Society that you have completed the requirement.

Questions concerning CME requirement can be directed to the Board or the Kansas Medical Society. The addresses of each are as follows:

The Kansas State Board of Healing Arts
503 Kansas Avenue
Topeka, KS 66603
Telephone: (913) 296-7413

The Kansas Medical Society
1300 Topeka Boulevard
Topeka, KS 66612
Telephone: (913) 235-2383

Each individual physician is responsible for recording his own hours of attendance at postgraduate courses. The credit hours should be recorded on the AMA Physician's Recognition Award application form when 150 hours are accumulated. The application form should then be mailed directly to the AMA, 535 North Dearborn, Chicago, IL 60610.

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Indications: For the symptomatic relief of bronchospastic conditions such as bronchial asthma, chronic bronchitis, and pulmonary emphysema.

Warnings: Do not administer more frequently than every 6 hours, or within 12 hours after rectal dose of any preparation containing theophylline or aminophylline. Do not give other compounds containing xanthine derivatives concurrently.

Precautions: Use with caution in patients with cardiac disease, hepatic or renal impairment. Concurrent administration with certain antibiotics, i.e. clindamycin, erythromycin, troleandomycin, may result in higher serum levels of theophylline. Plasma prothrombin and factor V may increase, but any clinical effect is likely to be small. Metabolites of guaifenesin may contribute to increased urinary 5-hydroxyindoleacetic acid readings, when determined with nitrosonaphtol reagent. Safe use in pregnancy has not been established. Use in case of pregnancy only when clearly needed.

Adverse Reactions: Theophylline may exert some stimulating effect on the central nervous system. Its administration may cause local irritation of the gastric mucosa, with possible gastric discomfort, nausea, and vomiting. The frequency of adverse reactions is related to the serum theophylline level and is not usually a problem at serum theophylline levels below 20 μg/ml.

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

CHANGE OF ADDRESS

Please notify the Kansas Medical Society of any changes in address

Help keep the mailing list up to date

The advertising in

THE JOURNAL

keeps you informed and helps pay the publishing costs!

When the local representative calls, tell him you saw his company's ad in your journal.

Medical School Codes

UNITED STATES

The following is a list of medical schools in the United States and Puerto Rico, existing and extinct, arranged in state order and showing the code number by which each school is designated in the geographical section of this Directory. Existing approved schools are listed in capital letters.

001 Alabama
001-02 UNIVERSITY OF ALABAMA SCHOOL OF MEDICINE, BIRMINGHAM
001-04 Birmingham Medical College
001-06 UNIVERSITY OF SOUTH ALABAMA SCHOOL OF MEDICINE, MOBILE

003 Arizona
003-01 UNIVERSITY OF ARIZONA COLLEGE OF MEDICINE, TUCSON

004 Arkansas
004-01 UNIVERSITY OF ARKANSAS SCHOOL OF MEDICINE, LITTLE ROCK
004-02 College of Physicians and Surgeons, Little Rock

005 California
005-01 Cooper Medical College, San Francisco
005-02 UNIVERSITY OF CALIFORNIA SCHOOL OF MEDICINE, SAN FRANCISCO
005-04 California Eclectic Medical College, Los Angeles
005-05 Hahnemann Medical College of the Pacific, San Francisco
005-06 UNIVERSITY OF SOUTHERN CALIFORNIA SCHOOL OF MEDICINE, LOS ANGELES
005-07 College of Physicians and Surgeons of San Francisco
005-08 Oakland College of Medicine and Surgery
005-09 College of Physicians and Surgeons, Los Angeles
005-11 STANFORD UNIVERSITY SCHOOL OF MEDICINE, PALO ALTO
005-12 LOMA LINDA UNIVERSITY SCHOOL OF MEDICINE, LOMA LINDA—LOS ANGELES
005-13 Pacific Medical College, Los Angeles
005-13 Pacific Medical College, Los Angeles
005-14 UNIVERSITY OF CALIFORNIA SCHOOL OF MEDICINE, LOS ANGELES
005-15 UNIVERSITY OF CALIFORNIA COLLEGE OF MEDICINE (CALIFORNIA COLLEGE OF MEDICINE), IRVINE
005-16 University of California, Irvine—California College of Medicine
005-17 University of California, Irvine—California College of Medicine
005-18 UNIVERSITY OF CALIFORNIA—SAN DIEGO, LA JOLLA
005-19 UNIVERSITY OF CALIFORNIA, SCHOOL OF MEDICINE, DAVIS
005-75 College of Osteopathic Physicians and Surgeons of Los Angeles

007 Colorado
007-02 UNIVERSITY OF COLORADO SCHOOL OF MEDICINE, DENVER

008 Connecticut
008-01 YALE UNIVERSITY SCHOOL OF MEDICINE, NEW HAVEN
008-02 UNIVERSITY OF CONNECTICUT SCHOOL OF MEDICINE, FARMINGTON

010 District of Columbia
010-01 GEORGE WASHINGTON UNIVERSITY SCHOOL OF MEDICINE, WASHINGTON
010-02 GEORGETOWN UNIVERSITY SCHOOL OF MEDICINE, WASHINGTON
010-03 HOWARD UNIVERSITY COLLEGE OF MEDICINE, WASHINGTON

011 Florida
011-02 UNIVERSITY OF MIAMI SCHOOL OF MEDICINE, MIAMI
011-03 UNIVERSITY OF FLORIDA COLLEGE OF MEDICINE, GAINESVILLE
011-04 UNIVERSITY OF SOUTH FLORIDA COLLEGE OF MEDICINE, TAMPA
011-05 BASIC MEDICAL SCIENCES PROGRAM, FLORIDA STATE UNIVERSITY, TALLAHASSEE

012 Georgia
012-01 MEDICAL COLLEGE OF GEORGIA, AUGUSTA
012-05 EMORY UNIVERSITY SCHOOL OF MEDICINE, ATLANTA
012-09 Georgia College of Eclectic Medicine and Surgery, Atlanta
012-10 Southern Medical College, Atlanta
012-11 Atlanta College of Physicians and Surgeons
012-12 Atlanta School of Medicine
012-18 Hospital Medical College Eclectic, Atlanta
012-19 Southern College of Medicine and Surgery, Atlanta

014 Hawaii
014-01 UNIVERSITY OF HAWAII SCHOOL OF MEDICINE, HONOLULU

016 Illinois
016-01 RUSH MEDICAL COLLEGE, CHICAGO
016-02 UNIVERSITY OF CHICAGO PRITZKER SCHOOL OF MEDICINE, CHICAGO
016-04 The Hahnemann Medical College and Hospital, Chicago
016-05 College of Medicine and Surgery, Chicago
016-06 NORTHWESTERN UNIVERSITY MEDICAL SCHOOL, CHICAGO
016-08 Bennett Medical College, Chicago
016-09 Northwestern University Woman's Medical School, Chicago
016-10 Chicago Homeopathic Medical College
016-11 UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE, CHICAGO
016-13 Harvey Medical College, Chicago
016-14 National Medical University, Chicago
016-15 Hering Medical College, Chicago
016-16 Jenner Medical College, Chicago
016-17 Illinois Medical College, Chicago
016-18 Dunham Medical College, Chicago
016-19 American Medical Missionary College, Battle Creek, Michigan & Chicago
016-22 Chicago College of Medicine and Surgery
016-23 Dearborn Medical College, Chicago
016-42 CHICAGO MEDICAL SCHOOL UNIVERSITY OF HEALTH SCIENCES, CHICAGO
016-43 LOYOLA UNIVERSITY STRITCH SCHOOL OF MEDICINE, MAYWOOD
016-44 The General Medical College, Chicago
016-45 SOUTHERN ILLINOIS UNIVERSITY SCHOOL OF MEDICINE, SPRINGFIELD
016-76 Chicago College of Osteopathy, Chicago

017 Indiana
017-05 Physiological Medical College of Indiana, Indianapolis
017-08 Medical College of Indiana, Indianapolis
017-09 Central College of Physicians and Surgeons, Indianapolis
017-17 Eclectic Medical College of Indiana, Indianapolis
017-18 Indiana Medical College, School of Medicine of Purdue University, Indianapolis
017-20 INDIANA UNIVERSITY SCHOOL OF MEDICINE, INDIANAPOLIS

018 Iowa
018-01 College of Physicians and Surgeons, Keokuk
018-03 UNIVERSITY OF IOWA COLLEGE OF MEDICINE, IOWA CITY
018-04 State University of Iowa College of Homeopathic Medicine, Iowa City
018-06 Drake University College of Medicine, Des Moines
018-08 Sioux City College of Medicine
018-10 Keokuk Medical College, College of Physicians and Surgeons
018-75 College of Osteopathic Medicine and Surgery, Des Moines

019 Kansas
019-02 UNIVERSITY OF KANSAS SCHOOL OF MEDICINE, KANSAS CITY
019-03 Kansas Medical College, Topeka
019-04 College of Physicians and Surgeons, Kansas City
019-07 Western Eclectic College of Medicine and Surgery, Kansas City

020 Kentucky

020-01 Kentucky School of Medicine, Louisville
020-02 UNIVERSITY OF LOUISVILLE SCHOOL OF MEDICINE, LOUISVILLE
020-04 Louisville Medical College
020-05 Hospital College of Medicine, Louisville
020-07 Louisville National Medical College
020-08 Southwestern Homeopathic Medical College and Hospital, Louisville
020-09 Kentucky University Medical Department, Louisville
020-11 Louisville and Hospital Medical College
020-12 UNIVERSITY OF KENTUCKY COLLEGE OF MEDICINE, LEXINGTON

021 Louisiana

021-01 TULANE UNIVERSITY SCHOOL OF MEDICINE, NEW ORLEANS
021-04 Flint Medical College of New Orleans University, New Orleans
021-05 LOUISIANA STATE UNIVERSITY SCHOOL OF MEDICINE, NEW ORLEANS
021-06 LOUISIANA STATE UNIVERSITY SCHOOL OF MEDICINE, SHREVEPORT

022 Maine

022-01 Bowdoin Medical School, Brunswick-Portland

023 Maryland

023-01 UNIVERSITY OF MARYLAND SCHOOL OF MEDICINE, BALTIMORE
023-03 College of Physicians and Surgeons of Baltimore
023-04 Baltimore Medical College
023-05 Woman's Medical College of Baltimore
023-06 Baltimore University School of Medicine
023-07 JOHNS HOPKINS UNIVERSITY SCHOOL OF MEDICINE, BALTIMORE
023-08 Atlantic Medical College, Baltimore
023-09 Maryland Medical College, Baltimore
023-11 Maryland College of Eclectic Medicine and Surgery, Baltimore

024 Massachusetts

024-01 HARVARD MEDICAL SCHOOL, BOSTON
024-05 BOSTON UNIVERSITY SCHOOL OF MEDICINE, BOSTON
024-06 College of Physicians and Surgeons, Boston
024-07 TUFTS UNIVERSITY SCHOOL OF MEDICINE, BOSTON
024-15 Middlesex University School of Medicine, Waltham
024-16 UNIVERSITY OF MASSACHUSETTS MEDICAL SCHOOL, WORCESTER
024-75 Massachusetts College of Osteopathy, Boston

025 Michigan

025-01 UNIVERSITY OF MICHIGAN MEDICAL SCHOOL, ANN ARBOR
025-05 University of Michigan Homeopathic Medical School, Ann Arbor
025-07 WAYNE STATE UNIVERSITY SCHOOL OF MEDICINE, DETROIT
025-08 Michigan College of Medicine and Surgery, Detroit
025-12 MICHIGAN STATE UNIVERSITY COLLEGE OF HUMAN MEDICINE, EAST LANSING
025-76 Michigan State University College of Osteopathic Medicine, East Lansing

026 Minnesota

026-04 UNIVERSITY OF MINNESOTA MEDICAL SCHOOL, MINNEAPOLIS
026-05 Minneapolis College of Physicians and Surgeons
026-07 UNIVERSITY OF MINNESOTA, SCHOOL OF MEDICINE, DULUTH
026-08 MAYO MEDICAL SCHOOL, ROCHESTER

027 Mississippi

027-01 UNIVERSITY OF MISSISSIPPI SCHOOL OF MEDICINE, JACKSON
207-02 Mississippi Medical College, Meridian

028 Missouri
028-01 Missouri Medical College, St. Louis
028-02 WASHINGTON UNIVERSITY SCHOOL OF MEDICINE, ST. LOUIS
028-03 UNIVERSITY OF MISSOURI SCHOOL OF MEDICINE, COLUMBIA
028-05 Homeopathic Medical College of Missouri, St. Louis
028-07 St. Louis College of Physicians and Surgeons
028-08 Kansas City Medical College
028-10 National University of Arts and Sciences Medical Department, St. Louis
028-20 University Medical College of Kansas City
028-22 Ensworth Medical College, St. Joseph
028-26 Kansas City Homeopathic Medical College
028-33 Eclectic Medical University, Kansas City
028-34 ST. LOUIS UNIVERSITY SCHOOL OF MEDICINE, ST. LOUIS
028-35 Southwest School of Medicine and Hospital, Kansas City
028-43 Kansas City College of Medicine and Surgery
028-44 Kansas City University of Physicians and Surgeons
028-45 Mid-West Medical College, Kansas City, See 028-43
028-46 UNIVERSITY OF MISSOURI SCHOOL OF MEDICINE, KANSAS CITY
028-78 Kansas City College of Osteopathy & Surgery
028-79 Kirksville College of Osteopathy & Surgery

030 Nebraska
030-04 Lincoln Medical College, Eclectic, Lincoln
030-05 UNIVERSITY OF NEBRASKA COLLEGE OF MEDICINE, OMAHA
030-06 CREIGHTON UNIVERSITY SCHOOL OF MEDICINE, OMAHA
030-07 Nebraska College of Medicine, Lincoln

031 Nevada
031-01 UNIVERSITY OF NEVADA SCHOOL OF MEDICAL SCIENCES, RENO

032 New Hampshire
032-01 DARTMOUTH MEDICAL SCHOOL, HANOVER

033 New Jersey
033-05 COLLEGE OF MEDICINE & DENTISTRY OF NEW JERSEY — NEW JERSEY MEDICAL SCHOOL, NEWARK
033-06 COLLEGE OF MEDICINE & DENTISTRY OF NEW JERSEY — RUTGERS MEDICAL SCHOOL, PISCATAWAY

034 New Mexico
034-01 UNIVERSITY OF NEW MEXICO SCHOOL OF MEDICINE, ALBUQUERQUE

035 New York
035-01 COLUMBIA UNIVERSITY COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK
035-03 ALBANY MEDICAL COLLEGE OF UNION UNIVERSITY, ALBANY
035-06 STATE UNIVERSITY OF NEW YORK AT BUFFALO, SCHOOL OF MEDICINE, BUFFALO
035-08 STATE UNIVERSITY OF NEW YORK COLLEGE OF MEDICINE, BROOKLYN
035-09 NEW YORK MEDICAL COLLEGE, NEW YORK
035-10 Bellevue Hospital Medical College, New York
035-11 New York Medical College and Hospital for Women, New York
035-13 Eclectic Medical College of The City of New York
035-15 STATE UNIVERSITY OF NEW YORK COLLEGE OF MEDICINE, SYRACUSE
035-19 NEW YORK UNIVERSITY SCHOOL OF MEDICINE, NEW YORK
035-20 CORNELL UNIVERSITY MEDICAL COLLEGE, NEW YORK
035-43 Fordham University School of Medicine, New York
035-45 UNIVERSITY OF ROCHESTER SCHOOL OF MEDICINE AND DENTISTRY, ROCHESTER
035-46 ALBERT EINSTEIN COLLEGE OF MEDICINE OF YESHIVA UNIVERSITY, NEW YORK

035-47 MOUNT SINAI SCHOOL OF MEDICINE OF THE CITY UNIVERSITY OF NEW YORK
035-48 STATE UNIVERSITY OF NEW YORK AT STONY BROOK SCHOOL OF MEDICINE, STONY BROOK

036 North Carolina
036-01 UNIVERSITY OF NORTH CAROLINA SCHOOL OF MEDICINE, CHAPEL HILL
036-03 Leonard Medical School, Raleigh
036-04 North Carolina Medical College, Charlotte
036-05 BOWMAN GRAY SCHOOL OF MEDICINE, WINSTON-SALEM
036-07 DUKE UNIVERSITY SCHOOL OF MEDICINE, DURHAM
036-08 EAST CAROLINA UNIVERSITY SCHOOL OF MEDICINE, GREENVILLE

037 North Dakota
037-01 UNIVERSITY OF NORTH DAKOTA SCHOOL OF MEDICINE, GRAND FORKS

038 Ohio
038-01 Medical College of Ohio, Cincinnati
038-02 Eclectic Medical College, Cincinnati
038-03 Starling Medical College, Columbus
038-06 CASE WESTERN RESERVE UNIVERSITY SCHOOL OF MEDICINE, CLEVELAND
038-08 Cincinnati College of Medicine and Surgery
038-09 Miami Medical College, Cincinnati
038-11 University of Wooster Medical Department, Cleveland
038-19 Toledo Medical College
038-21 Laura Memorial Woman's Medical College, Cincinnati
038-25 Ohio Medical University, Columbus
038-26 Cleveland-Pulte Medical College
038-40 OHIO STATE UNIVERSITY COLLEGE OF MEDICINE, COLUMBUS
038-41 UNIVERSITY OF CINCINNATI COLLEGE OF MEDICINE, CINCINNATI
038-42 Ohio State University College of Homeopathic Medicine, Columbus
038-43 MEDICAL COLLEGE OF OHIO AT TOLEDO, TOLEDO

039 Oklahoma
039-01 UNIVERSITY OF OKLAHOMA SCHOOL OF MEDICINE, OKLAHOMA CITY

040 Oregon
040-01 Willamette University Medical Department, Salem
040-02 UNIVERSITY OF OREGON MEDICAL SCHOOL, Portland

041 Pennsylvania
041-01 UNIVERSITY OF PENNSYLVANIA SCHOOL OF MEDICINE, PHILADELPHIA
041-02 JEFFERSON MEDICAL COLLEGE OF THOMAS JEFFERSON UNIVERSITY, PHILADELPHIA
041-07 MEDICAL COLLEGE OF PENNSYLVANIA, PHILADELPHIA
041-09 HAHNEMANN MEDICAL COLLEGE AND HOSPITAL, PHILADELPHIA
041-11 Medico-Chirurgical College of Philadelphia
041-12 UNIVERSITY OF PITTSBURGH SCHOOL OF MEDICINE, PITTSBURGH
041-13 TEMPLE UNIVERSITY SCHOOL OF MEDICINE, PHILADELPHIA
041-14 PENNSYLVANIA STATE UNIVERSITY COLLEGE OF MEDICINE, THE MILTON S. HERSHEY MEDICAL CENTER, HERSHEY
041-77 Philadelphia College of Osteopathic Medicine, Philadelphia

042 Puerto Rico
042-01 UNIVERSITY OF PUERTO RICO SCHOOL OF MEDICINE, SAN JUAN

043 Rhode Island
043-01 BROWN UNIVERSITY DIVISION OF BIOLOGICAL AND MEDICAL SCIENCES, PROVIDENCE

045 South Carolina
045-01 MEDICAL UNIVERSITY OF SOUTH CAROLINA COLLEGE OF MEDICINE, CHARLESTON

046 South Dakota
046-01 UNIVERSITY OF SOUTH DAKOTA SCHOOL OF MEDICINE, VERMILLION

047 Tennessee
047-01 University of Nashville Medical Department
047-05 VANDERBILT UNIVERSITY SCHOOL OF MEDICINE, NASHVILLE
047-06 UNIVERSITY OF TENNESSEE COLLEGE OF MEDICINE, MEMPHIS
047-07 MEHARRY MEDICAL COLLEGE SCHOOL OF MEDICINE, NASHVILLE
047-08 Memphis Hospital Medical College
047-09 Chattanooga Medical College
047-10 Lincoln Memorial University Medical Department, Knoxville
047-11 University of The South Medical Department, Sewanee
047-13 Knoxville Medical College
047-14 University of West Tennessee College of Medicine and Surgery, Memphis
047-15 College of Physicians and Surgeons, Memphis

048 Texas
048-02 UNIVERSITY OF TEXAS MEDICAL BRANCH, GALVESTON
048-03 Fort Worth School of Medicine
048-04 BAYLOR COLLEGE OF MEDICINE, HOUSTON
048-05 Physiological Medical College of Texas, Dallas
048-06 Southern Methodist University Medical Department, Dallas
048-07 Gate City Medical College, Dallas
048-08 College of Physicians and Surgeons, Dallas
048-12 UNIVERSITY OF TEXAS SOUTHWESTERN MEDICAL SCHOOL, DALLAS
048-13 UNIVERSITY OF TEXAS MEDICAL SCHOOL, SAN ANTONIO
048-14 UNIVERSITY OF TEXAS MEDICAL SCHOOL, HOUSTON
048-15 TEXAS TECH UNIVERSITY SCHOOL OF MEDICINE, LUBBOCK

049 Utah
049-01 UNIVERSITY OF UTAH COLLEGE OF MEDICINE, SALT LAKE CITY

050 Vermont
050-02 UNIVERSITY OF VERMONT COLLEGE OF MEDICINE, BURLINGTON

051 Virginia
051-01 UNIVERSITY OF VIRGINIA SCHOOL OF MEDICINE, CHARLOTTESVILLE
051-04 MEDICAL COLLEGE OF VIRGINIA HEALTH SCIENCES DIVISION OF VIRGINIA COMMONWEALTH UNIVERSITY, RICHMOND
051-06 University College of Medicine, Richmond
051-07 EASTERN VIRGINIA MEDICAL SCHOOL, NORFOLK

054 Washington
054-04 UNIVERSITY OF WASHINGTON SCHOOL OF MEDICINE, SEATTLE
054-15 Washington College of Physicians and Surgeons, Seattle

055 West Virginia
055-01 WEST VIRGINIA UNIVERSITY SCHOOL OF MEDICINE, MORGANTOWN

056 Wisconsin
056-02 Wisconsin College of Physicians and Surgeons, Milwaukee
056-03 Milwaukee Medical College
056-05 UNIVERSITY OF WISCONSIN MEDICAL SCHOOL, MADISON
056-06 MEDICAL COLLEGE OF WISCONSIN, MILWAUKEE

FOREIGN MEDICAL SCHOOL CODES

CANADA

060 Alberta
060-01 University of Alberta Faculty of Medicine, Edmonton
060-02 University of Calgary Faculty of Medicine

061 British Columbia
061-01 University of British Columbia Faculty of Medicine, Vancouver

062 Manitoba
062-01 University of Manitoba Faculty of Medicine, Winnipeg

063 Newfoundland
063-01 Memorial University of Newfoundland Faculty of Medicine, St. John's

064 Nova Scotia
064-01 Dalhousie University Faculty of Medicine, Halifax

065 Ontario
065-01 University of Toronto Faculty of Medicine, Toronto
065-02 University of Toronto Faculty of Medicine, Toronto
065-03 Medical Faculty of Trinity University, Toronto
065-05 Queen's University Faculty of Medicine, Kingston
065-06 University of Western Ontario Faculty of Medicine, London
065-09 University of Ottawa Faculty of Medicine, Ottawa
065-10 McMaster University Faculty of Medicine, Hamilton

067 Quebec
067-01 McGill University Faculty of Medicine, Montreal
067-02 University of Montreal Faculty of Medicine, Montreal
067-03 Laval University Faculty of Medicine, Quebec
067-04 Laval University Medical Faculty, Montreal
067-06 University of Sherbrooke, Faculty of Medicine, Sherbrooke

068 Saskatchewan
068-01 University of Saskatchewan College of Medicine, Saskatoon

OTHER FOREIGN

118 Afghanistan
118-01 Faculty of Medicine, Kabul University, Kabul

120 Albania
120-01 Faculty of Medicine, Tirana Shgiperi

121 Algeria
121- Algeria (Also see 125 Effective 1-1-71)
121-01 Faculte mixte de Medecine et de Pharmacie del'Universite d'Alger, Algiers

125 Algeria
125-01 Faculte mixte de Medecine et de Pharmacie del'Universite d'Alger, Algiers (121-01 Prior to 1-1-71)

132 Argentina
132-01 Facultad de Ciencias Medicas de la Universidad de Buenos Aires, Buenos Aires
132-02 Facultad de Ciencias Medicas de la Universidad Nacional de Cordoba, Cordoba, Cordoba
132-03 Facultad de Ciencias Medicas de la Universidad Nacional de la Plata, La Plata, Buenos Aires
132-04 Facultad de Ciencias Medicas, Farmacia y Ramos Menores de la Universidad Nacional del Litoral, Rosario, Santa Fe
132-05 Facultad de Medicina de la Universidad Nacional de Tucuman, Tucuman, Tucuman
132-06 Facultad de Ciencias Medicas de la Universidad Nacional de Cuyo, Mendoza, Mendoza

132-07 Facultad de Medicina de la Universidad del Salvador, Buenos Aires
132-08 Facultad de Medicina de la Universidad del Nordeste, Corrientes, Corrientes
132-09 Facultad de Medicina de la Universidad Catolica de Cordoba, Cordoba, Cordoba

143 Australia
143-01 Faculty of Medicine University of Adelaide, Adelaide, South Australia
143-02 Faculty of Medicine University of Melbourne, Parkville, Victoria
143-03 Faculty of Medicine University of Sydney, Sydney, New South Wales
143-05 Faculty of Medicine University of Queensland, Brisbane, Queensland
143-06 The University of Western Australia School of Medicine, Nedlands, Western Australia
143-07 Faculty of Medicine, University of New South Wales, Sydney, New South Wales
143-08 Faculty of Medicine Monash University, Clayton, Victoria

154 Austria
154-01 Medizinische Fakultat der Universitat Graz, Graz (407-27 from March 13, 1938 to June, 1945)
154-02 Medizinische Fakultat der Universitat Innsbruck, Innsbruck (407-28 from March 13, 1938 to June, 1945)
154-03 See 759-01
154-04 See 759-02
154-05 See 286-01
154-07 Medizinische Fakultat der Universitat Wien, Wien (407-26 from March 13, 1938 to June, 1945)

160 Bangladesh
160-01 Chittagung Medical College, Chittagong (704-10 Prior to 7-1-72)
160-02 Dacca Medical College, Dacca (704-03 Prior to 7-1-72)
160-04 Lytton Medical College, Mymensigh (704-12 Prior to 7-1-72)
160-05 Rajshahi Medical College, Rajshahi (704-11 Prior to 7-1-72)
160-06 Sylhet Medical College, Sylhet (704-13 Prior to 7-1-72)

165 Belgium
165-01 Faculte de Medecine et de Pharmacie Universite libra de Bruxelles, Bruxelles
165-02 Faculteit der Geneeskunde Rijksuniversiteit te Gent, Gent
165-03 Faculte de Medecine Universite de l'Etat a Liege, Liege
165-04 Faculte de Medecine de l'Universite catholique de Louvain, Louvain

176 Bolivia
176-01 Escuela de Medicina de la Universidad Mayor de San Andres, La Paz
176-02 Facultad de Ciencias Medicas de la Universidad Mayor Real y Pontificia de San Francisco Xavier de Chuquisaca, Sucre
176-03 Facultad de Medicina de la Universidad Mayor de San Simon, Cochabamba

187 Brazil
187-01 Faculdade de Medicina da Universidade de Bahia, Salvador, Bahia
187-02 Faculdade de Medicina de Porto Alegre, Porto Alegre, Rio Grande do Sul
187-03 Faculdade de Medicina da Universidade Federal do, Rio de Janeiro, Guanabara
187-04 Faculdade de Medicina da Universidade de Sao Paulo, Sao Paulo, Sao Paulo
187-05 Faculdade de Medicina do Paraiba, Joao Pessoa, Paraiba
187-06 Faculdade de Medicina da Universidade de Minas Gerais, Belo Horizonte, Minas Gerais
187-07 Faculdade de Medicina da Universidade do Recife Ciudade Universitaria-Engenho do Meio, Recife, Pernambuco
187-08 Faculdade de Medicina da Universidade de Parana, Curitiba, Parana
187-09 Faculdade de Ciencias Medicas, Rio de Janeiro, Guanabara
187-10 Faculdade de Medicina da Universidade do Ceara, Fortaleza, Ceara
187-11 Faculdade de Medicina de Ribeirao Preto Universidade de Sao Paulo, Ribeirao Preto, Sao Paulo
187-12 Escola Paulista de Medicina, Sao Paulo, Sao Paulo
187-13 Escola Fluminense de Medicina, Niteroi, Rio de Janeiro
187-14 Escola de Medicina do Espirito Santo, Vitoria, Espirito Santo
187-15 Faculdade de Ciencias Medicas de Maranhao, Sao Luis, Maranhao

187-16 Faculdade de Medicina da Universidade do Rio Grande do Norte, Natal, Rio Grande do Norte
187-17 Faculdade de Medicina de Santa Catarina, Florianapolis, Santa Catarina
187-18 Escola de Medicina e Cirurgia do Rio de Janeiro, Rio de Janeiro, Guanabara
187-19 Faculdade de Medicina de Sorocaba Pontificia Universidade Catolica de Sao Paulo, Sorocaba, Sao Paulo
187-20 Faculdade de Medicina Universidade do Para, Belem, Para
187-21 Faculdade de Ciencias Medicas de Pernambuco, Recife, Pernambuco
187-22 See 187-03
187-23 Faculdade de Medicina de Santa Maria, Universidade do Rio Grande do Sul, Santa Maria, Rio Grande do Sul, Brazil
187-24 Faculdade de Ciencias Medicas de Minas Gerais, Belo Harizonte, Minas Gerais
187-25 Faculdade Catolica de Medicina de Porto Alegre, Porto Alegre, Rio Grande do Sul
187-26 Faculdade de Ciencias Medicas do Parana, Curitiba, Parana
187-27 Faculdade de Medicina Fundacao da Cidade do Rio Grande do Sul, Rio Grande
187-28 Faculdade de Medicina do Triangulo Mineiro, Uberaba, Minas Gerais
187-29 Faculdade de Medicina, Universidade de Juiz de Fora, Juiz de Fora, Minas Gerais
187-30 Faculdade de Medicina, Universidade de Campinas, Campinas, Sao Paulo
187-31 Faculdade de Ciencias Medicas dos Hospitais da Santa Casa de Misericordia de Sao Paulo
187-32 Faculdade de Medicina da Universidade Federal de Goias, Goiania, Goias
187-33 Faculdade de Ciencias Medicas Fundacao Universidade De Brazilia, Brazilia DF Brazil
187-45 Guanabara Escola Medica do Rio Janeiro Sociedada Universitaria Gama Filho, Rio de Janeiro
187-46 Escola de Medicina e Saude Publicq Universidade Catolica Salvador, Salvador, Bahia

198 Bulgaria
198-01 Faculty of Medicine of the Cervenko Higher Institute of Medicine, Sofia
198-02 Pavlov Higher Institute of Medicine, Plovdiv

209 Burma
209-01 Faculty of Medicine University of Rangoon, Rangoon
209-02 Faculty of Medicine University of Mandalay, Mandalay
209-03 Medica College II Rangoon, Rangoon

215 Cambodia
215-01 Ecole Royal de Medicine Du Cambode, Phnompenh

220 Ceylon
220-01 Faculty of Medicine University of Ceylon, Colombo
220-02 Faculty of Medicine University of Ceylon, Peradeniya

231 Chile
231-01 Facultad de Medicina de la Universidad de Chile, Santiago
231-02 Facultad de Medicina de la Universidad de Concepcion, Concepcion
231-03 Facultad de Medicina de la Universidad Catolica de Chile, Santiago

242 China
242-03 Peiping Union Medical College, Peiping, Hopei (Extinct)
242-06 South China Medical College, Canton (Extinct)
242-07 National Hsiangya Medical College, Changsha, Hunan (Extinct)
242-09 St. John's University (Pennsylvania Medical School) Shanghai, Kiangsu (Extinct)
242-10 Cheeloo University School of Medicine, Tsinan, Shantung (Extinct)
242-13 Hackett Medical College for Women, Canton, Kwangtung (Extinct)
242-15 Women's Christian Medical College, Shanghai, Kiangsu (Extinct)
242-16 National Shanghai Medical College, Shanghai, Kiangsu
242-17 West China Union University College of Medicine and Dentistry, Chengtu, Szechuan
242-18 Army Medical College, Peking, Hopei (Extinct)

242-21 San Yat-sen Medical College of Lingnan University, Canton, Kwangtung
242-22 Aurora University Faculty of Medicine, Shanghai, Kiangsu (Extinct)
242-24 Tung Teh Medical College, Shanghai, Kiangsu (Extinct)
242-25 Army Medical College, Nanking, Kiangsu (Extinct)
242-26 National Central University College of Medicine, Nanking, Kiangsu (Extinct)
242-27 Tung Nan Medical College, Shanghai, Kiangsu (Extinct)
242-28 National Tung Chi University Faculty of Medicine, Shanghai, Kiangsu (Extinct)
242-29 National Kiangsu Medical College, Chingkiang, Kiangsu
242-30 National Kweiyang Medical College, Kweiyang, Kweichow
242-31 Lanchow University Medical College, Lanchow, Kansu (Extinct)
242-32 National Shantung University Medical College, Tsingtao, Shantung
242-33 Fukien Provincial Medical College, Foochow, Fukien
242-34 National Chung Cheng Medical College, Nanchang, Kiangsi (Extinct)
242-36 Hopei Provincial Medical College, Paoting, Hopei
242-37 Kuang Hua Medical School, Canton, Kwangtung
242-38 National Honan University Medical College, Kaifeng, Honan
242-39 Shansi University Medical College, Taiyuan, Shansi
242-40 National Northwest Medical College, Lanchow, Kansu
242-43 Chekiang Provincial Medical College, Hangchow, Chekiang
242-44 Harbin Medical University, Harbin, Sungkiang
242-45 Second Shanghai Medical College, Shanghai, Shanghai
242-46 Shantung Provincial Medical College, Tsinan, Shantung
242-47 National Peking University Medical College, Peking, Hopei
242-48 Tientsin Medical College, Tientsin, Hopei
242-49 Nanking University Medical College, Nanking, Kiangsu (Extinct)
242-50 National Chung Shan University (Sun Yat-sen) Medical College, Canton, Kwangtung
242-51 National Defense Medical Center School of Medicine, Shanghai, Kiangsu
242-52 Wuhan Medical College, Hankow, Hupeh
242-53 National Chang-Chun University, Chang Chun, Manchurio (Extinct)
242-54 North Kiangsu Medical College, Kiangsu, Nantung
242-55 Dairen Medical College, Liaotung, Dairen
242-56 Tungohi Medical College, Hupeh, Tsungnan, Wuchang
242-57 Anwhei Medical College, Anwhei, Hapei

243 China

243-16 National Shanghai Medical College, Shanghai, Kiangsu (242-16 Prior to 1-1-71)
243-17 West China University College Medical Dentistry Changtu (242-17 Prior to 1-1-71)
243-21 Sun Yat-Sen Medical College Lingnan University, Canton, Kwangtung (242-21 Prior to 1-1-71)
243-29 National Kiangsu Medical College, Chingkiang, Kiangsu (242-29 Prior to 1-1-71)
243-30 National Kweiyang Medical College, Kweiyang, Kweichow (242-30 Prior to 1-1-71)
243-32 National Shangtung University Medical College, Tsingtao, Shangtung (242-32 Prior to 1-1-71)
243-33 Fukien Provincial Medical College, Foochow, Fukien (242-33 Prior to 1-1-71)
243-36 Hopei Provincial Medical College, Paoting, Hopei (242-36 Prior to 1-1-71)
243-37 Kuang Hua Medical School, Canton, Kwangtung (242-37 Prior to 1-1-71)
243-38 National Honan University Medical College, Kaifeng, Honan (242-38 Prior to 1-1-71)
243-39 Shansi University Medical College, Taiyuan, Shansi (242-39 Prior to 1-1-71)
243-40 National Northwest Medical College, Lanchow, Chekiang (242-40 Prior to 1-1-71)
243-43 Chekiang Provincial Medical College, Hangchow, Chekiang (242-43 Prior to 1-1-71)
243-44 Harbin Medical University, Harbin, Sungkiang (242-44 Prior to 1-1-71)
243-45 Second Shanghai Medical College, Shanghai, Shanghai (242-45 Prior to 1-1-71)
243-46 Shantung Provincial Medical College, Tsinan, Shantung (242-46 Prior to 1-1-71)

243-47 National Peking University Medical College, Peking, Hopei (242-47 Prior to 1-1-71)
243-48 Tientsin Medical College, Tientsin, Hopei (242-48 Prior to 1-1-71)
243-50 National Chung Shan University Medical College, Canton, Kwangtung (242-50 Prior to 1-1-71)
243-51 National Defense Medical Center, School of Medicine, Shanghai, Kiangsu (242-51 Prior to 1-1-71)
243-52 Wuhan Medical College, Hankow, Hupeh (242-52 Prior to 1-1-71)
243-54 North Kiangsu Medical College, Kiangsu, Nantung (242-54 Prior to 1-1-71)
243-55 Dairen Medical College, Liaotung, Dairen (242-55 Prior to 1-1-71)
243-56 Tungchi Medical College, Hupeh, Tsungnan, Wuchang (242-56 Prior to 1-1-71)
243-57 Anwhei Medical College, Anwhei, Hopei (242-57 Prior to 1-1-71)
243-61 Kwangsi Provincial Medical College, Kweilin, Kwangsi

244 Taiwan
244 Taiwan (Formosa) effective 1-1-71
244-01 Kaohsiung (Takau) Medical College, Kaohsiung (385-01 Prior to 1-1-71)
244-02 College of Medicine National Taiwan University, Taipei (385-02 Prior to 1-1-71)
244-03 National Defense Medical Center, Taipei (385-03 Prior to 1-1-71)
244-04 Taipei Medical College, Taipei (385-04 Prior to 1-1-71)
244-05 China Medical College, Taichung (385-05 Prior to 1-1-71)
244-06 Chung-Shan Medical College, Taichung

264 Colombia
264-01 Facultad de Medicina de la Universidad Nacional de Colombia Ciudad Universitaria, Bogota, Cundinamarca
264-02 Facultad de Medicina de la Universidad de Cartagena, Cartagena, Bolivar
264-03 Facultad de Medicina de la Universidad de Antioquia, Medellin, Antioquia
264-04 Facultad de Medicina de la Pontificia Universidad Javeriana, Bogota, Cundinamarca
264-05 Facultad de Medicina de la Universidad del Valle, Cali, Valle
264-06 Facultad de Medicina de la Universidad de Caldas, Manizales, Caldas
264-07 Facultad de Medicina de la Universidad del Cauca, Popayan, Cauca
264-10 Facultad de Medicina Colegio Mayor de Nuestro Senora del Rosario, Bogota

266 Zaire
266 Formerly Congo
266-01 Faculte de Medecine de L'Universite Lovanium, Kimuenza, Leopoldville

270 Costa Rica
270-01 Facultad de Medicina Universidad de Costa Rica, San Jose, Costa Rica

275 Cuba
275-01 Facultad de Medicina de la Universidad de la Habana, La Habana
275-02 Escuela de Medicina Universidad de Oriente, Santiago

286 Czechoslovakia
286-01 Deutsche Universitat Medizinische Fakultat, Praha (154-05 Prior to 1919)
286-02 Fakulta Vseobeckeno Lekarstyi Karlova Universita, Katerinska, Praha
286-03 Lekarska Fakulta Universita Komenskeho, Bratislava
286-04 Lekarska Fakulta Universita J. E. Purkyne, Brno
286-05 Lekarska Fakulta Palackeho Universita, Lidicka, Olomouc
286-06 Lekarska Fakulta Safarikova Universita, Kosice

297 Denmark
297-01 Det Laegevidenskabelige Fakultet Kobenhavns Universitet, Kobenhavn
297-02 Det Laegevidenskabelige Fakultet Aarhus Universitet, Aarhus

308 Dominican Republic
308-01 Facultad de Medicina de la Universidad de Santo Domingo, Ciudad, Trujillo
308-02 Escuela de Medicina Universidad, Nacional Pedro Henriquez, Urena, Santo Domingo

315 East Africa (Uganda)
315 (Also see 905 UGANDA (Effective 1-1-71))
315-01 Faculty of Medicine Makerere College, University College of East Africa, Kampala, Uganda

319 Ecuador
319-01 Facultad de Ciencias Medicas de la Universidad Central, Quito
319-02 Facultad de Ciencias Medicas de la Universidad de Cuenca, Cuenca
319-03 Facultad de Ciencias Medicas de la Universidad de Guayaquil, Guayaquil

330 Egypt (United Arab Republic)
Also see 915 United Arab Republic (Effective 1-1-71)
330-01 Ibrahim Pasha University Faculty of Medicine, Cairo
330-02 Kasr-el-Aini-Faculty of Medicine Cairo University, Cairo (Formerly Fouad First University Faculty of Medicine)
330-03 Faculty of Medicine Alexandria University, Alexandria
330-04 Abbasis Faculty of Medicine University of Ein Shams, Cairo

341 El Salvador
341-04 Facultad de Medicina Universidad Nacional del Salvador, San Salvador

352 England
(Also see 917 United Kingdom—England—Wales (Effective 1-1-71)
352-01 The Medical School University of Birmingham, Birmingham
352-02 Faculty of Medicine University of Bristol, Bristol
352-03 Cambridge University Medical School, Cambridge
352-04 University of Newcastle-upon-Tyne, The Medical School (King's College in the University of Durham) (Prior to August 1, 1963)
352-05 School of Medicine University of Leeds, Leeds
352-06 Faculty of Medicine University of Liverpool, Liverpool
352-07 University of London Faculty of Medicine, London, includes the following Medical Schools:
a. University College Hospital
b. King's College Hospital Medical School
c. The Medical College of St. Bartholomew's Hospital
d. St. Thomas's Hospital Medical School
e. Westminster Medical School
f. Guy's Hospital Medical School
g. St. George's Hospital Medical School
h. The London Hospital Medical College
i. The Middlesex Hospital Medical School
j. Charing Cross Hospital Medical School
k. Royal Free Hospital School of Medicine
l. St. Mary's Hospital Medical School
352-08 Faculty of Medicine Victoria University of Manchester, Manchester
352-09 Oxford University Medical School, Oxford
352-10 Faculty of Medicine University of Sheffield, Sheffield
352-11 Registrable Qualifications granted by English Conjoint Board (Royal College of Surgeons of England/Royal College of Physicians of London)
352-17 Registrable Qualification granted by Society of Apothecaries of London

363 Estonia
363-01 Tartu Ulikooli-Faculte de Medecine de l'Universite de Tartu, Tartu (Extinct)

366 Ethiopia
366-01 Haile Selassie I University, Addis Ababa

368 Fiji
368-01 Central Medical School, Suva

374 Finland
374-01 Laaketieteellinen Tiedekunta Helsingin Yliopisto, Helsinki
374-02 Laaketieteellinen Tiedekunta Turun Yliopisto, Turku
374-03 Faculty of Medicine University of Oulu, Oulu

385 Formosa (Taiwan)

Also see 244 Taiwan (Effective 1-1-71)

385-01 Kaohsiung (takau) Medical College, Kaohsiung
385-02 College of Medicine National Taiwan University, Taipei
385-03 National Defense Medical Center, Taipei
385-04 Taipei Medical College, Taipei
385-05 China Medical College, Taichung

396 France

396-01 Faculte mixte de Medecine et de Pharmacie de l'Universite de Bordeaux, Bordeaux, Gironde
396-02 Faculte mixte de Medecine et de Pharmacie de l'Universite de Lille, Lille, Nord
396-03 Faculte mixte de Medecine et de Pharmacie Universite de Lyon, Lyon, Rhone
396-04 Faculte de Medecine de Montpellier Rue de l'Ecole-de-Medecine, Montpellier, Herault
396-05 Faculte de Medecine de l'Universite de Nancy, Nancy, Meurthe-et-Moselle
396-06 Faculte de Medecine de l'Universite de Paris, Paris, Seine
396-07 Faculte mixte de Medecine et de Pharmacie de l'Universite de Toulouse, Toulouse, Haute-Garonne
396-08 Faculte de Medecine de l'Universite de Strasbourg, Strasbourg, Bas-Rhin (407-18 Prior to 1918)
396-10 Ecole nationale de Medecine et de Pharmacie Universite de Caen, Rouen, Seine-Maritime
396-11 Faculte mixte de Medecine et de Pharmacie de Marseille Universite d'Aix-Marseille, Marseille, Bouches-du-Rhone
396-15 Faculte mixte de Medecine et de Pharmacie, Nantes, Loire-Atlantique
396-16 Ecole national de Medecine et de Pharmacie Universite de Besancon, Besancon, Doubs
396-17 Faculte mixte de Medecine et de Pharmacie Universite de Clermont-Ferrand, Clermont-Ferrand, Puy-de-Dome
396-18 Ecole nationale de Medecine et de Pharmacie Universite de Grenoble, Grenoble, Isere
396-19 Ecole nationale de Medecine et de Pharmacie de Pharmacie de Tours, Tours, Indre-et Loire
396-27 Ecole nationale de Medecine et de Pharmacie Reims, Marne

407 Germany

Also see 048-409—East and West Germany (Effective 1-1-71)

407-01 Friedrich-Wilhelms-Universitat Medizinische Fakultat, Berlin, Prussia
407-02 Medizinische Fakultat der Rheinischen Friedrich-Wilhelms-Universitat, Bonn, Nordrhein-Westfalen
407-03 Schlesische-Friedrich-Wilhelms-Universitat Medizinische Fakultat, Breslau, Prussia
407-04 Medizinische Fakultat der Universitat Erlangen, Erlangen, Bayern
407-05 Medizinische Fakultat der Albert-Ludwigs-Universitat, Freiburg-Im-Breisgau, Baden-Wurttemberg
407-06 Medizinische Fakultat der Justus-Liebig-Universitat, Giessen, Hessen
407-07 Medizinische Fakultat der Georg-August-Universitat, Gottingen, Niedersachsen
407-08 Medizinische Fakultat der Universitat Greifswald, Greifswald
407-09 Medizinische Fakultat der Martin-Luther Universitat, Halle
407-10 Medizinische Fakultat der Universitat Heidelberg, Heidelberg, Baden-Wurttemberg
407-11 Medizinische Fakultat der Friedrich-Schiller-Universitat, Jena
407-12 Medizinische Fakultat der Christian-Albrechts-Universitat, Kiel, Schleswig-Holstein
407-13 Albertus-Universitat Medizinische Fakultat, Konigsberg, Prussia
407-14 Universitat Leipzig Medizinische Fakultat, Saxony
407-15 Medizinische Fakultat der Phillipps-Universitat, Marburg/Lahn, Hessen
407-16 Medizinische Fakultat der Ludwig Maximiliams-Universitat, Munchen, Bayern
407-17 Medizinische Fakultat der Universitat Rostock, Rostock
407-18 Kaiser-Wilhelms-Universitat Medizinische Fakultat, Strassburg (Under French control since 1918. See 396-08)

407-19 Medizinische Fakultat der Eberhard-Karls-Universitat, Tubingen, Baden-Wurttemberg
407-20 Medizinische Fakultat der Julius-Maximiliams-Universitat, Wurzburg, Bayern
407-21 Medizinische Fakultat der Universitat Hamburg, Hamburg, Hamburg
407-22 Medizinische Fakultat der Universitat Koln, Koln, Nordrhein-Westfalen
407-23 Medizinische Fakultat der Johann-Wolfgang-Goethe-Universitat, Frankfurt-Am-Main, Hessen
407-24 Medizinische Fakultat der Westfalischen Landes-Universitat, Munster, Nordrhein-Westfalen
407-25 Medizinische Akademie in Dusseldorf, Dusseldorf, Nordrhein-Westfalen
407-26 Medizinische Fakultat der Universitat Wien, Wien (154-07 Prior to March 13, 1938 and since June 1945)
407-27 Karl-Franzens-Universitat Medizinische Fakultat, Graz, Graz (154-01 Prior to March 13, 1938 and since June 1945)
407-28 Leopold-Franzens-Universitat Medizinische Fakultat, Innsbruck, Innsbruck (154-02 Prior to March 13, 1938 and since June 1945)
407-29 United Hungarian University Medical School, Halle
407-30 Medizinische Fakultat der Humboldt-Universitat, Berlin
407-32 Medizinische Fakultat der Johannes-Gutenberg-Universitat, Mainz, Rheinland-Platz
407-33 Medizinische Fakultat der Freien Universitat Berlin, Berlin
407-34 Medizinische Fakultat der Universitat des Saarlandes, Homburg, Saarland
407-35 Klinikum Essen Der Ruhr Universitat Bochun, Essen
407-36 Medizinische Akademie Luebeck II Fakultat Medizinische Christian-Albrechts Universitat Kiel, Luebeck

408 Germany East

408-08 Medizinische Fakultat de Universitat, Greifswald, Greifswald (407-08 Prior to 1-1-71)
408-09 Medizinische Fakultat Martin Luther Universitat, Halle (407-09 Prior to 1-1-71)
408-11 Medizinische Fakultat Friedrich-Schiller Universitat, Jena (407-11 Prior to 1-1-71)
408-14 Medizinische Fakultat Karl-Marx Universitat, Leipzig (formerly 407-14 Universitat Leipzig Prior to 1-1-71)
408-17 Medizinische Fakultat Universitat, Rostock, Rostock (407-17 Prior to 1-1-71)
408-30 Medizinische Fakultat Humboldt Universitat, Berlin (407-30 Prior to 1-1-71)

409 Germany West

409-02 Medizinische Fakultat der Rheinischen Friedrich-Wilhelms-Universitat, Bonn, Nordrhein-Westfalen (407-02 Prior to 1-1-71)
409-04 Medizinische Fakultat Universitat Erlangen, Erlangen, Bayern (401-04 Prior to 1-1-71)
409-05 Medizinische Fakultat Albert-Ludwigs-Universitat Freiburg IM Breisgau (407-05 Prior to 1-1-71)
409-06 Medizinische Fakultat Justus-Liebig Universitat Giessen, Hessen (407-06 Prior to 1-1-71)
409-07 Medizinische Fakultat George August-Universitat Gottingen, Niedersachen (407-07 Prior to 1-1-71)
409-10 Medizinische Fakultat Universitat Heidelberg, Baden-Wurttemberg (407-10 Prior to 1-1-71)
409-12 Medizinische Fakultat Christian-Albrechts Universitat Liel (407-12 Prior to 1-1-71)
409-15 Medizinische Fakultat Philipps Universitat, Marburg Lahn Hessen (407-15 Prior to 1-1-71)
409-16 Medizinische Fakultat Ludwig Maximiliams-Universitat Munchen, Bayern (407-16 Prior to 1-1-71)
409-19 Medizinische Fakultat Eberhard Karls Universitat, Tubingen (407-19 Prior to 1-1-71)
409-20 Medizinische Fakultat Julius Maximiliams Universitat, Wurzburg, Bayern (407-20 Prior to 1-1-71)
409-21 Medizinische Fakultat Universitat Hamburg, Hamburg, Hamburg (407-21 Prior to 1-1-71)

409-22 Medizinische Fakultat Universitat Koln, Nordrhein-Westfalen (407-22 Prior to 1-1-71)
409-23 Medizinische Fakultat Johann-Wolfgang-Goethe Universitat Frankfurt-Am-Main Hessen (407-23 Prior to 1-1-71)
409-24 Medizinische Fakultat Westfalische Landes Universitat, Munster, Nordrhein-Westfalen (407-24 Prior to 1-1-71)
409-25 Medizinische Akademie Dusseldorf, Dusseldorf, Nordrheim-Westfalen (407-25 Prior to 1-1-71)
409-32 Medizinische Fakultat Johannes Gutenberg Universitat, Mainz, Rheinland-Platz (407-32 Prior to 1-1-71)
409-33 Medizinische Fakultat Freien Universitat, Berlin, Berlin (407-33 Prior to 1-1-71)
409-34 Medizinische Fakultat Universitat, Saarlandes, Homburg, Saarland (407-34 Prior to 1-1-71)
409-35 Klinikum Essen Der Ruhr Universitat Bochum, Essen (407-35 Prior to 1-1-71)
409-36 Medizinische Akademie Luebeck II Fakultat Medizinische Christian-Albrechts Universitat Kiel, Luebeck (407-36 Prior to 1-1-71)
409-39 Medizinische Fakultat der Rheinisch-Westfalen Tech Hochschule Aachen Nordrhein-Westfalen

Goa (See India)

418 Greece
418-01 Faculty of Medicine National University of Athens, Athens
418-02 Faculty of Medicine University of Thessaloniki, Thessaloniki

429 Guatemala
429-01 Facultad de Ciencias Medicas de la Universidad de San Carlos de Guatemala, Guatemala

440 Haiti
440-01 Faculte de Medecine et de Pharmacie de l'Universite d'Haiti, Port-Au-Prince

451 Honduras
451-01 Facultad de Medicine y Cirugia de la Universidad Nacional Autonoma de Honduras, Tegucigalpa

462 Hong Kong
462-01 Faculty of Medicine University of Hong Kong, Hong Kong

473 Hungary
473-01 Orvosi Fakultas Tudomanyegyetem, Budapest
473-02 Orvosi Fakultas Szegedi Orvostudomanyi Egyetem, Szeged
473-03 Orvosi Fakultas Pecsi Tudomanyegyetem, Pecs
473-04 Orvosi Fakultas Tudomanyegyetem, Debrecen
473-05 Magyar Kiralyi Tudomamyegyetem Orvostudomany Fakultas, Koloszvar (Extinct)

484 Iceland
484-01 Laeknadeild Haskola Islands, Reykjavik

495 India (Goa)
495-01 University of Bombay, Affiliated Medical Colleges are:
a. Grant Medical College Bombay University, Bombay, Maharashtra
b. Seth Gorhandas Sunderdas Medical College Bombay University, Bombay, Maharashtra
495-02 Medical College Calcutta University, Calcutta, West Bengal
495-03 Medical College Punjab University, Amritsar, Affiliate Medical College is:
a. Lady Hardinge Medical College for Women and Hospital for Women and Children, New Delhi, Delhi
495-04 Madras Medical College Madras University, Madras, Madras
495-05 King George's Medical College Lucknow University, Lucknow, Uttar Pradesh
495-08 Christian Medical College Punjab University, Ludhiana, Punjab
495-09 St. John's Medical College, Bangalore, Mysore (before June 1966: Government Medical College, Mysore University, Mysore)

495-11 Andhra Medical College Andhra University, Visakhapatnam, Andhra
495-12 Sarojini Najdu Medical College Agra University, Agra, Uttar Pradesh
495-13 Sriram Chandra Bhanj Medical College Utkal University, Cuttack, Orissa
495-14 Ayurvedio Medical College, Benares (Constituent College of Benares Hindu University)
495-15 Prince of Wales Medical College Patna University, Patna, Bihar
495-16 Stanley Medical College Madras University, Madras, Madras
495-17 Topiwala National Medical College, Bombay, Maharashtra
495-18 Assam Medical College Gauhati University, Dibrugarh, Assam
495-19 Medical College and Hospital Nagpur University, Nagpur, Maharashtra
495-20 Mahatma Gandhi Memorial Medical College, Indore, Madhya, Pradesh
495-21 Osmania Medical College Osmania University, Hyderabad, Andhra
495-22 B. J. Medical College Gujarat University, Ahmedabad, Gujarat
495-23 Medical College Baroda University, Baroda, Gujarat
495-24 Darbhanga Medical College Bihar University, Laheriasarai, Bihar
495-25 Escola Medico-Cirurgica de Goa, Goa
495-27 Christian Medical College, Vellore, Madras
495-28 Byramjee Jeejeebhoy Medical College, Poona, Maharashtra
495-29 Government Medical College Punjab University, Patiala, Punjab
495-30 Sawai Man Singh Medical College Rajasthan University, Jaipur, Rajasthan
495-31 Medical College Kerala University, Trivandrum, Kerala
495-32 Nilratan Sircar Medical College Calcutta University, Calcutta, West Bengal
495-33 Government Medical College, Mysore University (Before June 1966 Bangalore Medical College, Mysore University, Bangalore)
495-34 Gajra Rajo Medical College Vikram University, Gwalior Madhya Pradesh
495-35 Karnatak Medical College Karnatak University, Hubli, Mysore
495-36 All-India Institute of Medical Sciences, New Delhi, Delhi
495-37 Kasturba Medical College Karnatak University, Manipal, Mysore
495-38 R. G. Kar Medical College Calcutta University, Calcutta, West Bengal
495-39 Calcutta National Medical Institute Calcutta University, Calcutta, West Bengal
495-40 O.H.N. Medical College Gujarat University, Navrangpura, Bombay
495-41 G.S.V. Memorial Medical College Lucknow University, Kampur, Uttar Pradesh
495-42 Madurai Medical College, Madurai, Madras
495-43 Arya Medical School Punjab State Medical Faculty, Ludhiana, Punjab
495-44 Medical College Kerala University, Calicut, Kerala
495-45 Maulana Azad Medical College, New Delhi, Delhi
495-46 Bankura Sammilani Medical College Calcutta University, Bankura, West Bengal
495-47 Medical College Jabalpur University, Jabalpur, Madhya Pradesh
495-48 M.P. Shah Medical College Gujarat University, Jamnagar, Gujarat
495-49 Gandhi Medical College Vikram University, Bhopai, Madhya Pradesh
495-50 Guntur Medical College Andhra University, Guntur, Andhra
495-51 Medical College, Srinagar, Jammu and Kashmir
495-52 Bangalore Medical College, Bangalore, Mysore
495-53 Medical College, Pondicherry
495-54 Rajendra Medical College, Ranchi, Bihar
495-55 Sardar Patel Medical College, Bikaner, India
495-56 Medical College and Civil Hospital Marathwada University, Aurangabad, Maharashtra
495-57 Kakatiya Medical College, Warangal, Andhra Pradesh
495-58 Shree Rangaraya Memorial Medical College, Andhra University, Kakinada, Andhra
495-59 Kilpauk Medical College, Madras, Madras
495-60 Jawaharlal Institute of Post Graduate Medicine, Pondicherry
495-61 Medical College, Chingleput, Madras
495-62 Kurnool Medical College Tiraipati University, Kurnool, Andhra
495-63 Medical College, Kotayam, Kerala
495-64 Shree J.S.M.G.A. Medical College, Nadiad, Bombay
495-65 Gandhi Medical College, Osmania University, Hyderabad, Andhra
495-66 Medical College, Thanjavin, Madras

495-67 Motilai Nehru Medical College, Allahabad, Uttar Pradesh
495-68 College Medical Sciences Banaras Hindu University Varanasi, Uttar Pradesh
495-69 Medical College, Rohtak, Punjab
495-70 Sri Venkateswara Medical College Tirupati University, Tirupati, Andhra
495-71 Medical College, Osmania University, Warangal, Andhra
495-72 Medical College Bellary, Mysore
495-73 Armed Forces Medical College Hospital, Poona, Maharash
495-74 Medical College Udaipur, Rajasthan
495-75 Medical College Jameshedpur, Bihar
495-75 Medical College Jamshedpur, Bihar
495-77 Medical College, Aligarh, Uttar Pradesh
495-78 Medical College, Gauhati, Assam
495-79 Burla Medical College-Hospital Utkal University, Sambalpur, Orissa
495-80 Medical College, Alleppey, Kerala
495-81 Medical College, Rajasthan University, Bikamer, Rajasthan
495-82 Medical College, Miraj, Maharashtra
495-90 Himachal Pradesh Medical College, Simla Himachal, Pradesh
495-94 Coimbatore Medical College, University of Madras, Coimbatore, Madras
495-95 Medical College, Tirunelveli, Madras
495-96 Municipal Medical College, Seion University, Bombay (Lokmanya T.M.M. College, Bombay Maharashitra)
495-97 Dr. Vaishampayan Memorial Medical College, Shivaji University, Shalopur, Maharashitra
495-98 Jawaharial Nehru Medical College, Karnatak University, Belgaum, Mysore
495-99 J.J.M. Medical College Mysore University, Devangere, Mysore

496 India
496-01 Hyderabad Karnatak Education Society's Medical College, Karnatak University, Galbarga, Mysore
496-02 Medical College, Ajmer, Rajasthan

506 Indonesia
506-01 Faculty of Medicine University of Indonesia, Djakarta
506-02 Faculty of Medicine Airlangga Airlangga University, Surabaya
506-03 Faculty of Medicine Gadjah Mada University, Djokjakarta
506-04 Faculty of Medicine University of North Sumatra, Medan
506-05 Faculty of Medicine Padjadjaran University, Bandung
506-11 Faculty of Medicine, Malang, Malang

517 Iran
517-01 Faculty of Medicine University of Teheran, Teheran
517-03 Faculty of Medicine, Tabriz
517-04 Faculty of Medicine, Meshed
517-05 Faculty of Medicine, Shiraz
517-06 Faculty of Medicine, Isfahan
517-07 Faculty of Medicine, Ohwaz
517-08 School of Medicine National University of Iran, Teheran

528 Iraq
528-01 Faculty of Medicine Baghdad University, Baghdad
528-02 College of Medicine Baghdad University, Mosul

539 Ireland
539-01 Faculty of Medicine Queen's University of Belfast, Belfast (Also see 918-01 effective 1-1-71)
539-02 National University of Ireland, Constituent Colleges are:
a. Faculty of Medicine University College, Dublin
b. Faculty of Medicine University College, Cork
c. Faculty of Medicine, Galway
539-03 School of Physic Trinity College University of Dublin, Dublin
539-06 Registrable Qualification granted by the Royal College of Physicians of Ireland and Royal College of Surgeons in Ireland
539-11 Registrable Qualification granted by Apothecaries' Hall of Dublin

550 Israel
550-01 The Hebrew University-Hadassah Medical School, Jerusalem
550-02 University of Tel-Aviv, Tel-Aviv

561 Italy
561-01 Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna
561-02 Facolta di Medicina e Chirurgia dell'Universita di Cagliari, Cagliari
561-03 Facolta di Medicina e Chirurgia dell'Universita di Milano, Milano
561-04 Facolta di Medicina e Chirurgia dell'Universita di Catania, Catania
561-06 Facolta di Medicina e Chirurgia dell'Universita di Firenze, Firenze
561-07 Facolta di Medicina e Chirurgia dell'Universita di Genova, Genova
561-08 Facolta di Medicina e Chirurgia dell'Universita di Messina, Messina
561-09 Facolta di Medicina e Chirurgia dell'Universita di Modena, Modena
561-10 Facolta di Medicina e Chirurgia dell'Universita di Napoli, Napoli
561-11 Facolta di Medicina e Chirurgia dell'Universita di Padova, Padova
561-12 Facolta di Medicina e Chirurgia dell'Universita di Palermo, Palermo
561-13 Facolta di Medicina e Chirurgia dell'Universita di Parma, Parma
561-14 Facolta di Medicina e Chirurgia dell'Universita di Pavia, Pavia
561-15 Facolta di Medicina e Chirurgia dell'Universita di Perugia, Perugia
561-16 Facolta di Medicina e Chirurgia dell'Universita di Pisa, Pisa
561-17 Facolta di Medicina e Chirurgia dell'Universita di Roma, Roma
561-18 Facolta di Medicina e Chirurgia dell'Universita di Sassari, Sassari
561-19 Facolta di Medicina e Chirurgia dell'Universita di Siena, Siena
561-20 Facolta di Medicina e Chirurgia dell'Universita di Torino, Torino
561-21 Facolta di Medicina e Chirurgia dell'Universita di Bari, Bari
561-22 Facolta di Medicina e Chirurgia dell'Universita di Ferrara, Ferrara
561-23 Facolta di Medicina-Chirurgia dell'Universita Catolica de Sacro Cuoro Di Milano, Roma

563 Ivory Coast
563-01 Ecole De Medecine University D'Abidjan, Abidjan

566 Jamaica (see West Indies)
566 Jamaica (Also see 950 West Indies)
566-01 Faculty Medicine University College, West Indies, Kingston, Jamaica (950-01 Prior to 1-1-71)

572 Japan
572-01 Faculty of Medicine Kyoto University, Kyoto, Kyoto
572-03 Faculty of Medicine University of Tokyo, Tokyo, Tokyo
572-04 School of Medicine Nagoya National University, Nagoya, Aichi
572-05 School of Medicine Chiba University, Chiba, Chiba
572-06 Faculty of Medicine Kanazawa University, Kanazawa, Ishikawa
572-07 School of Medicine Kumamoto University, Kumamoto, Kumamoto
572-08 Faculty of Medicine Nagasaki University, Nagasaki, Nagasaki
572-09 Okayama University Medical School, Okayama, Okayama
572-10 Faculty of Medicine Tohoku University, Sendai, Miyagi
572-11 Tokyo Medical College (Nippon Ikadaigaku) Hongo, Tokyo (Extinct)
572-12 Faculty of Medicine Kyushu University, Fukuoka, Fukuoka
572-13 Kyoto Prefectural University of Medicine, Kyoto, Kyoto
572-15 School of Medicine Niigata University, Niigata, Niigata
572-16 Faculty of Medicine Osaka University, Osaka, Osaka
572-17 Tokyo Charity Association Medical College, Tokyo, Tokyo (Extinct)
572-18 Tokyo Women's Medical College, Tokyo, Tokyo
572-20 School of Medicine Keio Gijuku University, Tokyo, Tokyo
572-24 Okinawa Medical Institute, Naha, Okinawa (Extinct)
572-25 Japan University Medical Department (Nippon Daigaku Ikadajgaku) Kanda, Tokyo
572-26 Showa Medical School, Tokyo, Tokyo
572-27 Medical College of Toho University, Tokyo, Tokyo
572-28 Faculty of Medicine Nihon University, Tokyo, Tokyo
572-29 Faculty of Medicine Hokkaido University, Sapporo, Hokkaido
572-30 Kobe Medical College, Kobe, Hyogo

572-31 Yamaguchi Prefectural University of Medicine, Ube Yamaguchi
572-32 School of Medicine Yokohama Municipal University, Yokohama, Kanagawa
572-33 School of Medicine Gunma University, Maebashi, Gunma
572-34 Faculty of Medicine Mie Prefectural University, Tsu, Mie
572-35 School of Medicine Tokyo Medical and Dental University, Tokyo, Tokyo
572-36 Hiroshima University Medical School, Hiroshima, Hiroshima
572-37 Tokyo Jikei-Kai School of Medicine, Tokyo, Tokyo
572-38 Nippon Medical School, Tokyo, Tokyo
572-39 Iwate Medical University, Morioka, Iwate
572-40 Faculty of Medicine Juntendo University, Tokyo, Tokyo
572-41 Faculty of Medicine Shinshu University, Matsumoto, Nagano
572-42 Kansai Medical College, Moriguchi, Osaka
572-43 Faculty of Medicine Tottori University, Yonago, Tottori
572-44 Faculty of Medicine Hirosaki University, Hirosaki, Aomari
572-45 Faculty of Medicine Kagoshima University, Kagoshima, Kagoshima
572-46 Gifu Prefectural Medical College, Gifu, Gifu
572-47 Medical School Nagoya City University, Nagoya, Aichi
572-48 Osaka City Medical School, Osaka, Osaka
572-49 Tokyo Medical College, Toyko, Tokyo
572-50 Faculty of Medicine Kurume University, Kurume, Fukuoka
572-51 Osaka Medical College, Takatsuki, Osaka
572-52 School of Medicine Tokushima University, Tokushima, Tokushima
572-53 Sapporo Medical College, Sapporo, Hokkaido
572-54 Wakayama Prefectural Medical College, Wakayama, Wakayama
572-55 Nara Prefectural Medical College, Kashiwabara, Nara
572-56 Fukushima Prefectural Medical College, Fukushima, Fukushima

582 Korea (North)
582-05 Pyong Yang Medical College, Pyong Yang (583-05 Prior to 1-1-71)
582-07 Ham Heung Medical School, Ham Heung (583-07 Prior to 1-1-71)

583 Korea (South)
583-01 Severance Medical College Yonsei University, Seoul
583-02 College of Medicine Seoul National University, Seoul
583-03 Korea University Medical College (Formerly Woo Sok University Medical College) (Formerly Soo Do Medical College, Seoul)
583-04 College of Medicine Kyong-Puk National University, Taegu
583-05 Pyon Yang Medical College, Pyong Yang, North Korea (582-05 as of 1-1-71)
583-06 College of Medicine Chun Nam National University, Kwangiu
583-07 Ham Heung Medical School, Ham Heung, North Korea (582-07 as of 1-1-71)
583-08 College of Medicine Ewha Women's University, Seoul
583-09 College of Medicine Pusan National University, Pusan
583-10 College of Medicine Catholic University, Seoul
583-12 College of Medicine Kyonghue University, Seoul

590 Laos
590-01 Ecole Royale de Medecine Du Laos, Vientiane

594 Latvia
594-01 Latvijas Universitate Medicinas Fakultate, Riga (Extinct)

605 Lebanon
605-01 Medical School American University of Beirut, Beirut
605-02 Faculte Francaise de Medecine et de Pharmacie de l'Universite Saint-Joseph, Beyrouth

616 Lithuania
616-01 Vytauta Didziojo Universiteto Medicinos Fakelteto, Kaunas (Extinct)

624 Malaysia
624-01 University of Malaya, Kuala, Lumpur

627 Malta
627-01 Faculty of Medicine and Surgery Royal University of Malta, Valetta

638 Manchuria
638-01 Manchuria Medical College, Mukden (Extinct)
638-02 Mukden Medical College, Mukden (Extinct)

649 Mexico
649-01 Facultad de Medicina de la Universidad Nacional Autonoma de Mexico, Mexico
649-02 Facultad de Medicina de la Universidad de Nuevo Leon, Monterrey, Nuevo Leon
649-03 Facultad de Medicina de la Universidad de Guadalajara, Guadalajara, Jalisco
649-04 Facultad de Medicina de la Universidad Autonomo de San Luis Potosi, San Luis Potosi, San Luis Potosi
649-05 Escuela Medico Militar Mexico, D.F.
649-06 Facultad de Medicina de la Universidad Nacional del Sureste, Merida, Yucatan
649-07 Facultad de Medicina de la Universidad Michoacana de San Nicolas de Hidalgo, Morelia, Michoacan
649-08 Escuela Libre de Homeopatia de Mexico, Mexico
649-09 Escuela Libre de Homeopatia del Estado de Puebla, Puebla, Puebla (Extinct)
649-10 Escuela de Medicina y Cirugia de la Universidad "Benito Juarez" de Oaxaca, Oaxaca
649-11 Instituto Libre Homeopatia de Mexico, Mexico (Extinct)
649-14 Facultad de Medicina de la Universidad Autonoma de Guadalajara, Guadalajara, Jalisco
649-15 National Homeopatia Mexico School, Mexico City (Extinct)
649-17 Escuela Nacional de Medicina Homeopatia del Instituto Politecnico Nacional, Mexico
649-18 Facultad de Medicina de la Universidad Autonoma de Puebla, Puebla, Puebla
649-19 Escuela de Medicina de la Universidad de Tamaulipas, Tampico, Tamaulipas
649-20 Escuela de Medicina "Miguel Aleman" de la Universidad Veracrusana, Veracruz, Veracruz
649-21 Escuela de Medicina del Instituto Cientifico y Literaio Autonoma del Estado de Hildalgo, Pachuaca, Hidalgo
649-22 Escuela de Medicina de la Universidad Juarez del Estado de Durango, Durango, Durango
649-23 Escuela de Medicina de la Universidad de Coahuila, Torreon, Coahuila
649-24 Escuela de Medicina de la Universidad de Campeche, Campeche, Campeche
649-25 Escuela de Medicina de Leon Universidad de Guanajuato, Leon, Guanajuato
649-26 Escuela Superior de Medicina Rural de Instituto Politecnico Nacional, Mexico, D.F.
649-27 Facultad de Medicina de la Universidad de Chihuahua, Chihuahua, Chihuahua
649-28 Escuela de Medicina de la Universidad Autonoma del Estado de Mexico, Toluca

660 Netherlands
660-01 Faculteit der Geneeskunde Universiteit Van Amsterdam, Amsterdam
660-02 Medische Faculteit Rijksuniversiteit te Groningen, Groningen
660-03 Medische Faculteit Rijksuniversiteit te Leiden, Leiden
660-04 Faculteit der Geneeskunde Rijksuniversiteit te Utrecht, Utrecht
660-05 Faculteit der Geneeskunde Roomsch-Katholieke Universiteit te Nijmegen, Nijmegen
660-06 Faculteit der Geneeskunde Vrije Universiteit te Amsterdam, Amsterdam
660-07 Medische Faculteit Rotterdam, Rotterdam

671 New Zealand
671-01 Medical School University of Otago, Dunedin

682 Nicaragua
682-01 Facultad de Medicina y Cirugia de la Universidad Nacional de Nicaragua, Leon

690 Nigeria
690-01 Faculty of Medicine University College, Ibadan
690-02 Medical School University of Lagos, Lagos

692 North Viet-Nam
(Also see 938 Viet-Nam North Effective 1-1-71)
692-01 Faculte mixte de Medicine et de Pharmacie Universite de Hanoi, Hanoi

693 Norway
693-01 Medisinske Fakultet Universitetet i Oslo, Oslo
693-02 Medisinske Fakultet Universitetet i Bergen, Bergen

704 Pakistan
(See also 160 Bangladesh)
704-01 King Edward Medical College, Lahore, West Pakistan
704-02 Dow Medical College, Karachi, Federal Capital Area
704-03 Dacca Medical College, Dacca, East Pakistan
704-04 Nishtar Medical College, Multan, West Pakistan
704-05 Punjab Medical School, Lahore, West Pakistan (Extinct)
704-06 Fatima Jinnah Medical College for Women, Lahore, West Pakistan
704-07 C.M.S. Medical School, Hyderabad, West Pakistan (Extinct)
704-08 Liaquat Medical College, Hyderabad, West Pakistan
704-09 Khyber Medical College, Peshawar, North-West Frontier Province
704-10 Chittagong Medical College, Chittagong, East Pakistan (160-01 after 7-1-72)
704-11 Rajshahi Medical College, Rajshahi, East Pakistan (160-05 after 7-1-72)
704-12 Lytton Medical College, Mymensingh, East Pakistan (160-04 after 7-1-72)
704-13 Sylhet Medical College, Sylhet, East Pakistan (160-06 after 7-1-72)

715 Panama
715-01 Facultad de Medicina de la Universidad de Panama, Panama

726 Paraguay
726-01 Facultad de Medicina de la Universidad Nacional de Asuncion, Asuncion

737 Peru
737-01 Facultad de Medicina de San Fernando de la Universidad Nacional Mayor de San Marcos, Lima
737-03 Facultad de Medicina de la Universidad de la Libertad, Trujillo
737-05 Facultad de Medicina de la Universidad Nacional de San Agustin, Arequipa
737-06 Facultad de Medicina "Cayetano Heredia" de la Universidad Peruana de Ciencias Medicas y Biologicas, Lima
737-08 Facultad de Medicina Universidad Nacional de San Luis Gonzaga de Ica, Ica

748 Philippines
748-01 Faculty of Medicine and Surgery University of Santo Tomas, Manila
748-02 College of Medicine University of the Philippines, Manila
748-05 Afable College of Medicine, Manila (Extinct)
748-07 College of Medicine Manila Central University, Manila
748-08 Institute of Medicine Far Eastern University, Manila
748-09 College of Medicine Southwestern University, Cebu City
748-10 College of Medicine University of the East, Quezon City
748-11 College of Medicine Cebu Institute of Technology, Cebu City

759 Poland
759-01 Akademia Medyczna, Krakow (154-03 Prior to 1919)
759-02 Uniwersytet Jana Kazimierza Wydzial Lekarski, Lwow (154-04 Prior to 1919)
759-03 Akademia Medyczna Wydzial Lekarski, Warszawa
759-04 Akademia Medyczna Wydzial Lekarski, Poznan
759-05 Uniwersytet Stefana Batorego Wydzial Lekarski, Wilno (Extinct)
759-06 Marie Curie Sklodowska University Faculty of Medicine, Lublau
759-07 Akademia Medyczna Gdansk
759-08 Pomorska Akademia Medyczna im.gen. K.Swierczewskiego, Szczecin
759-09 Akademia Medyczna, Lodz
759-10 Akademia Medyczna, Wroclaw
759-11 Akademia Medyczna, Bialystock
759-12 Slaska Akademia Medyczna im. Ludwika Warynskiego, Zabrze

770 Portugal
770-01 Faculdade de Medicina de la Universidade de Coimbra, Coimbra
770-02 Faculdade de Medicina de la Universidade de Lisboa, Lisboa
770-03 Faculdade de Medicina de la Universidade do Porto, Porto

775 Rhodesia
775-01 Faculty of Medicine University College of Rhodesia and Myasaland, Salisbury, Southern Rhodesia

781 Romania
781-01 Institutul de Medicina si Farmacie, Bucuresti
781-02 Institutul de Medicina si Farmacie, Iasi
781-03 Institutul de Medicina si Farmacie, Cluj
781-04 Institutul de Medicina, Timisoara
781-05 Institutul de Medicina si Farmacie, Tirgu-Mures

803 Scotland
(Also see 919 United Kingdom-Scotland effective 1-1-71)
803-01 Faculty of Medicine University of Aberdeen, Aberdeen
803-02 University of St. Andrews School of Medicine, Dundee
803-03 Faculty of Medicine University of Edinburgh, Edinburgh
803-04 School of Medicine of the Royal Colleges, Edinburgh (Extinct)
803-05 Faculty of Medicine University of Glasgow, Glasgow
803-06 Anderson College of Medicine, Glasgow (Extinct)
803-09 Registrable Qualification granted by Scottish Conjoint Board
803-16 Polish School of Medicine, Edinburgh (Extinct)

820 Senegal
820-01 Faculte mixte de Medicine et de Pharmacie, Universite de Dakar, Dakar

825 Singapore
825-01 Faculty of Medicine University of Malaya, Singapore

836 South Africa
836-01 Medical School University of the Witwatersrand, Johannesburg
836-02 Faculty of Medicine University of Cape Town, Cape Town
836-03 Faculty of Medicine University of Pretoria, Pretoria
836-04 Faculty of Medicine University of Stellenbosch, Bellville
836-05 Faculty of Medicine University of Natal, Durban

840 South Viet-Nam
(Also see 941 Viet-Nam South)
840-01 Faculte mixte de Medecine et de Pharmacie Universite de Saigon, Saigon

847 Spain
847-01 Facultad de Medicina de la Universidad de Barcelona, Barcelona
847-02 Universidad de Sevilla
a. Facultad de Medicina de Cadiz, Cadiz
b. Facultad de Medicina de la Universidad de Sevilla, Sevilla
847-03 Facultad de Medicina de la Universidad de Granada, Granada
847-04 Facultad de Medicina de la Universidad de Madrid, Madrid
847-05 Facultad de Medicina de la Universidad de Santiago de Compostela, Santiago De Compostela
847-06 Facultad de Medicina de la Universidad de Zaragoza, Zaragoza
847-08 Facultad de Medicina de la Universidad de Valencia, Valencia
847-09 Facultad de Medicina de la Universidad de Valladolid, Valladolid
847-10 Facultad de Medicina de la Universidad e Salamanca, Salamanca
847-11 Facultad de Medicina de la Universidad Catolica de Navarra, Pomplona

848 Sudan
848-01 Faculty of Medicine University of Khartoum, Khartoum (849-01 Prior to 1-1-71)

849 Sudan
(Also see 848 SUDAN Effective 1-1-71)
849-01 Faculty of Medicine, University of Khartoum, Khartoum

850 Surinam
850-01 Geneeskundige School, Paramaribo

858 Sweden
858-01 Medicinska Fakulteten Universitetet i Lund, Lund

858-02 Kungl. Karolinska Mediko-Kirurgiska Institutet, Stockholm
858-03 Medicinska Fakulteten Universitetet i Uppsala, Uppsala
858-04 Kungl. Medicinska Hogskolan, Umea
858-05 Medicinska Fakulteten Goteborgs Universitetet, Goteborg

869 Switz erland
869-01 Medizinische Fakultat der Universitat Basel, Basel
869-02 Medizinische Fakultat der Universitat Bern, Bern
869-04 Faculte de Medecine de l'Universite de Geneva, Geneve
869-05 Faculte de Medecine de l'Universite de Lausanne, Lausanne
869-07 Medizinische Fakultat der Universitat Zurich, Zurich

875 Syria
875-01 Faculty of Medicine Damascus University, Damascus

Taiwan (See Formosa)

891 Thailand
891-01 Faculty of Medicine at Chulalongkorn Hospital University of Medical Sciences, Bangkok
891-02 Faculty of Medicine at Siriraj Hospital University of Medical Sciences, Thonburi
891-03 Faculty of Medicine at Chiengmai Hospital University of Medical Sciences, Chiengmai

902 Turkey
902-01 Tip Fakultesi Istanbul Universitesi, Istanbul
902-03 Tip Fakultesi Ankara Universitesi, Ankara
902-04 Tip Fakultesi Ege Universitesi, Izmir
902-05 Haceteppe Universitesi Faculty of Medicine, Ankara

905 Uganda
905-01 Faculty of Medicine Makerere College University College of East Africa, Kampala (315-01 Prior to 1-1-71)

913 Union of Soviet Socialist Republics
913-01 First Leningrad Medical Institute (I.P. Pavlov Institute), Leningrad
913-02 Voronez Medical Institute, Voronez
913-03 Kazan' Medical Institute, Kazan'
913-04 Harkov Medical Institute, Harkov
913-05 Kiev Medical Institute, Kiev
913-06 First Moscow Order-of-Lenin Medical Institute, Moscow
913-07 Odessa Medical Institute, Odessa
913-08 Tomsk Medical Institute, Tomsk
913-09 Leningrad Institute of Sanitation and Hygiene, Leningrad
913-10 Dnepropetrovsk Medical Institute, Dnepropetrovsk
913-11 Saratov Medical Institute, Saratov
913-12 Rostov Medical Institute, Rostov-on-Don
913-13 Crimean Medical Institute, Simferopol
913-14 Irkutsk Medical Institute, Irkutsk
913-15 Second Moscow Medical Institute, Moscow
913-17 Military Medical Academy, Leningrad (Extinct)
913-18 Vinnica Medical Institute, Vinnica
913-19 Azerbaijan Medical Institute, Baku
913-20 Imperatorskii Novorossiiksi Universitet, Odessa (Extinct)
913-21 Taskent Medical Institute, Taskent
913-22 Kuban Medical Institute, Krasnodar
913-23 Tbilisi Medical Institute, Tbilisi
913-24 Stalino Institute of Medicine, Stalino (Extinct)
913-26 Samarkand Medical Institute, Samarkand
913-28 Third Medical Institute, Leningrad
913-29 Kazakh Medical Institute, Alma-Ata
913-30 Askabad Medical Institute, Askabad
913-31 Daghestan Medical Institute, Mahac-Kala
913-32 Byelorussia Medical Institute, Minsk

913-33 Vitebsk Medical Institute, Vitebsk
913-34 Frunze Medical Institute, Frunze
913-35 Kursk Medical Institute, Kursk
913-36 Gor 'Kij Medical Institute Gor'Ku (Gorki)
913-37 Kujbysev Medical Institute, Kujbysev
913-38 Erevan Medical Institute, Erevan, Armenian SSR
913-69 Leningrad Institute Paediatrics, Leningrad, Russian SSR
913-92 Faculty of Medicine P Lumumbra Friendship University, Moscow

915 United Arab Republic
915-01 Ibrahim Pasha University Faculty Medicine, Cairo (330-01 Prior to 1-1-71)
915-02 Kas-el Aini-Faculty Medicine Cairo University, Cairo (330-02 Prior to 1-1-71)
915-03 Faculty Medicine Alexandria University, Alexandria (330-03 Prior to 1-1-71)
915-04 Abbasis Faculty Medicine University Ein Shams, Cairo (330-04 Prior to 1-1-71)
915-05 Faculty of Medicine Assyut University, Assyut
915-08 Faculty of Medicine El Azhar University, Cairo

917 United Kingdom—England—Wales
917-01 The Medical School University Birmingham, Birmingham (352-01 Prior to 1-1-71)
917-02 Faculty of Medicine University Bristol, Bristol (352-02 Prior to 1-1-71)
917-03 Cambridge University Medical School, Cambridge (352-03 Prior to 1-1-71)
917-04 University of Newcastle-Upon-Tyne, The Medical School (352-04 Prior to 1-1-71)
917-05 School of Medicine University of Leeds, Leeds (352-05 Prior to 1-1-71)
917-06 Faculty of Medicine University of Liverpool (352-06 Prior to 1-1-71)
917-07 University of London Faculty Medicine, London (352-07 Prior to 1-1-71)
a. University College Hospital
b. Kings College Hospital Medical School
c. The Medical College of St. Bartholomew's Hospital
d. St. Thomas Hospital Medical School
e. Westminster Medical School
f. Guys Hospital Medical School
g. St. Georges Hospital Medical School
h. The London Hospital Medical College
i. The Middlesex Hospital Medical School
j. Charing Cross Hospital Medical School
k. Royal Free Hospital School Medicine
l. St. Mary's Hospital Medical School
917-08 Faculty Medical Victoria University Manchester, Manchester (352-08 Prior to 1-1-71)
917-09 Oxford University Medical School, Oxford (352-09 Prior to 1-1-71)
917-10 Faculty Medical University Sheffield, Sheffield (352-10 Prior to 1-1-71)
917-11 Registrable Qualification granted by English Conjoint Board, Royal College of Surgeons of England/Royal College of Physicians (352-11 Prior to 1-1-71)
917-17 Registrable Qualification granted — Society of Apothecaries of London (352-17 Prior to 1-1-71)
917-18 The Welsh National School Medicine, Cardiff (946-01 Prior to 1-1-71)

918 United Kingdom—Northern Ireland
918-01 Faculty Medicine Queens University Belfast, Belfast (539-01 Prior to 1-1-71)

919 United Kingdom—Scotland
919-01 Faculty Medicine University Aberdeen, Aberdeen (803-01 Prior to 1-1-71)
919-02 University St. Andrews School Medicine, Dundee (803-02 Prior to 1-1-71)
919-03 Faculty Medicine University Edinburgh, Edinburgh (803-03 Prior to 1-1-71)
919-05 Faculty Medicine University Glasgow, Glasgow (803-05 Prior to 1-1-71)
919-09 Registrable Qualification granted by Conjoint Scottish Board (803-09 Prior to 1-1-71)

925 Uruguay
924-01 Facultad de Medicina de la Universidad de la Republica, Montevideo

935 Venezuela

935-01 Facultad de Medicina Universidad Central de Venezuela Cludad Universitaria, Caracas
935-02 Facultad de Medicina Universidad de Los Andes, Merida
935-03 Facultad de Ciencias Medicas Universidad Nacional del Zulia, Maracaibo
935-04 Facultad de Medicina Universidad de Carabobo, Valencia

938 Viet-Nam North

938-01 Faculte mixte de Medicine et de Pharmacie Universite de Hanoi, Hanoi (692-01 Prior to 1-1-71)

941 Viet-Nam South

941-01 Faculte mixte de Medicine et de Pharmacie Universite de Saigon, Saigon (840-01 Prior to 1-1-71)

946 Wales

(Also see 917 United Kingdom-England-Wales Effective 1-1-71)
946-01 The Welsh National School of Medicine, Cardiff

950 West Indies (Jamaica)

(Also see 566 Jamaica Effective 1-1-71)
950-01 Faculty of Medicine University College of the West Indies, Kingston, Jamaica

957 Yugoslavia

957-01 Medicinski Fakultet Sveucilista u Zagrebu, Zagreb
957-02 Medicinski Fakultet Univerziteta u Beogradu, Beograd
957-03 Fakulteta za Splosno Medicino in Stomatologijo, Ljubljana
957-04 Medicinski Fakultet Univerziteta vo Skoplje, Skoplje
957-05 Medicinski Fakultet (Medical Faculty) Rijeka
957-07 Medicinski Fakultet, Novi Sad
957-08 Medicinski Fakultet Univerziteta U Sarajevo, Sarajevo

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100

No information available as to scientific medical education.

200

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BICHLMEIER, FRANKLIN G, KC
BIERI, PETER V, LAWRENCE
BIERLEIN, KENNETH J, PITTSBURG
BIERMANN, ALOYSIUS H, GARDEN PLAIN
BIERMANN, HENRY J, WICHITA
BIERMANN, WILLIAM J, WICHITA
BIGGS, DENNIS, ABILENE
BIGLER, F CALVIN, GARDEN CITY
BIGONGIARI, LAWRENCE R, KC
BILLINGS, THOMAS, MCPHERSON
BILLINGSLEY, THAD H, SHAWNEE MISSION
BILLINGSLEY JR, JOHN A, SHAWNEE MISSION
BINYON, KERNIE W, WICHITA
BISHOP, FRANCIS E, SHAWNEE MISSION
BISHOP, RODNEY L, LAWRENCE
BITTENBENDER, LEE R, LAWRENCE
BLACK, CYRIL V, PRATT
BLACK, JAMES F, HONOLULU,HI
BLACK, WILLIAM L, AURORA,CO
BLACKBURN, ROBERT W, COUNCIL GROVE
BLAIR, T RICHARD, LAWRENCE
BLAKE, HENRY S, TOPEKA
BLANK, JOHN N, HUTCHINSON
BLAYLOCK, HOYT C, WICHITA
BLETZ, DONALD B, SHAWNEE MISSION
BLISS, JOY V, OLATHE
BLOOD, MARY J, WICHITA
BLOOM, L THEIL, KINGMAN
BOESE, KENNETH M, MANHATTAN
BOGGAN, MICHAEL D, KC
BOLES, J MICHAEL, SHAWNEE MISSION
BOLES, R DALE, DODGE CITY
BOLINGER, ROBERT E, KC
BOLLMAN, CHARLES S, JUNCTION CITY
BOLT, MICHAEL, WICHITA
BOLTON, VICTOR E, KC
BONO, ROGER C, WICHITA
BORGENDALE, LLEWELLYN V, WAMEGO
BORKLUND, MAURICE K, PARSONS
BORRA, MARIO J, HUTCHINSON
BOS, NORMAN C, HUTCHINSON
BOSILJEVAC, FRED N, SHAWNEE MISSION
BOSILJEVAC, JOSEPH E, WICHITA
BOSSE, FRANK K, ATCHISON
BOSWELL, H CRAIG, KC
BOUDET, ROBERT A, KC,MO
BOUDREAUX, VELTIN J, HALSTEAD
BOWEN, CLOVIS W, TOPEKA
BOWEN JR, HARRY J, TOPEKA
BOWMAN, HAROLD S, WICHITA
*BOYD, SPENCER H, TOPEKA
BOYD, Z REX, WICHITA
BOYDEN, MARY S, LAWRENCE
BOYER, ROBERT E, KINGMAN
BOYLE, HUGH H, WICHITA
BRACKETT JR, CHARLES E, KC
BRADEN, BILL L, WAMEGO
BRADLEY, HOWARD R, EMPORIA
BRADLEY, J RODERICK, GREENSBURG
BRADY, CHARLES S, ATCHISON
BRAKE, DAVID, WICHITA
BRALEY, JACK A, O.D., ARKANSAS CITY
BRANDSTED, ERNEST C, MCPHERSON
BRANSON, VERNON L, LAWRENCE
BRAUN, EDWARD W, FORT SCOTT
BRAUN, THOMAS G, WICHITA
BRAUN, WILLIAM T, PITTSBURG
BRAUN III, WILLIAM T, WICHITA
BRAUNSDORF, ROBERT L, TOPEKA
BRAY, AVIS PAGE, CONCORDIA
BRAY, EDEAN, LAWRENCE
*BRECKBILL, DAVID L, WICHITA
BRENNER, WILLIAM R, LARNED
BRETHOUR, LESLIE J, JUNCTION CITY
BREWER, MARSHALL A, ULYSSES
BRIAN, ROBERT M, EL DORADO
BRIBACH, EUGENE J, ATCHISON
BRIDGENS, JAMES G, SHAWNEE MISSION
BRIDWELL, RUSSELL E, TOPEKA
BRILLHART, MAXINE T, KC
BRINTON, EDWARD S, WICHITA
BRITO, RAUL E, WICHITA
BROCKHOUSE, JOHN P, EMPORIA
BROOKER, ROBERT M, TOPEKA
BROSIUS, FRANK C, WICHITA
BROUCEK, FRANCIS J, TOPEKA
BROWN, ALEX L, CHAUTAUQUA,NY
BROWN, C EVERETT, STAFFORD
BROWN, C REIFF, GREAT BEND
BROWN, DAVID J, WICHITA
BROWN, FRED E, ST MARYS
BROWN, HARWIN J, WINFIELD
BROWN, PAUL W, OLATHE
BROWN, ROBERT L, WICHITA
BROWN, ROBERT M, MANHATTAN
BROWN, ROBERT O, ATCHISON
BROWN, RONALD C, WICHITA
BROWN, RONALD L, WICHITA
BROWN, VAL J, WICHITA
BROWN, VIRGIL E, SABETHA
BROWN, WILLIAM R, SHAWNEE MISSION
BROWN-SANDERS, CAROLINE, LEES SUMMIT,MO

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BROWNING, WILLIAM H, WICHITA
BROWNRIGG, RICHARD L, DODGE CITY
BRUMMETT, RICHARD R, SALINA
BRUNER, STEVEN C, LAWRENCE
BRUNER JR, KENNETH W, WICHITA
BRUNGARDT, BERNARD A, SALINA
BRUNO, JAMES W, GARDEN CITY
BRYAN, EMERY C, ERIE
BRYANT, HOMER L, COFFEYVILLE
BUAKHAM, CHANA, KC
BUBB, STEPHEN K, SHAWNEE MISSION
BUBECK, RALPH W, WICHITA
BUCHNER, DAVID M, SHAWNEE MISSION
BUCK JR, BEN H, WICHITA
BUCK JR, HENRY W, LAWRENCE
BUDETTI, JOSEPH A, WICHITA
BULA, RALPH E, HAYS
BULLOCK, HAROLD O, INDEPENDENCE
BUNKER JR, HERBERT L, JUNCTION CITY
BURDICK, BRUCE M, MANHATTAN
BURGER, J DALE, HUTCHINSON
BURGER, PAUL B, SHAWNEE MISSION
BURGER, WILLIAM E, KC
BURGESON, FRANK G, EMPORIA
BURGESS, ARTHUR P, OSWEGO
BURKE, JAMES J, FORT SCOTT
BURKE, JOSEPH V, ATCHISON
BURKET JR, GEORGE E, KC
BURKMAN, REUBEN J, CHANUTE
BURNETT, A DEAN, HALSTEAD
BURNEY, WILLIAM W, WICHITA
BURPEE, JAMES F, WICHITA
BUSCH, ANTHONY B, DODGE CITY
BUSTAMANTE, SERGIO A, TUCSON, AZ
BUTCHER, ARCHIE W, ABILENE
BUTCHER, THOMAS P, EMPORIA
BUTH, DENNIS K, WICHITA
BUTIN, J WALKER, WICHITA
BUTT, MUHAMMAD, CONCORDIA
BUTTON, JESSE H, LAWRENCE

C

CABLE, THOMAS M, VERSAILLES,KY
CAEDO, CARMELITA D, LIBERAL
CAIN, IVAN W, SHAWNEE MISSION
CALDERON, JAIME, SHAWNEE MISSION
CALDERWOOD, WILLIAM A, SALINA
CALIENDO JR, DANIEL J, WICHITA
CALKINS, LARRY L, SHAWNEE MISSION
CALKINS, W GRAHAM, KC
CAMERON, WILLIAM J, KC
CAMPBELL, EDWARD G, EMPORIA
CAMPBELL, FRANCES S, NEWTON
CAMPBELL, GARLAND L, ARKANSAS CITY
CAMPBELL, WILLIAM H, COFFEYVILLE
CAMPION, WOODROW M, LIBERAL
CANELLOS, JAMES, GOESSEL
CAPODTH, L WAYNE, TOPEKA
CAPPER, STANLEY L, WICHITA
CARBAUGH, KENNETH W, BELLA VISTA,AR
CARDUFF, JAY J, SHAWNEE MISSION
CAREY, LARRY J, SHAWNEE MISSION
CARLETON, RICHARD C, CLAY CENTER
CARLSON, EARL V, HAYS
CARMAN, DEAN R, QUINTER
*CARNAHAN, ROBERT L, WICHITA
CARNEY, MYRTLE S, FT WORTH,TK
CARPENTER, PAUL R, KC
CARPER, IVAN H, NEWTON
CARPER, OWEN E, NEWTON
CARRASCO, LENORE C, SHAWNEE MISSION
CARREAU, ERNEST P, WICHITA
CARRO, F AURELIO, WINFIELD
CARSON, RICHARD CARLYLE, PITTSBURG
CARTER, MACK A, WICHITA
CARTER JR, ROBERT P, KC
*CARTWRIGHT, LYLE B, WICHITA
CARVER, LARRY A, TOPEKA
CASADY, GILBERT N, HUTCHINSON
CASEY, JAMES, HUTCHINSON
CASEY, JOHN J, WICHITA
CASHMAN JR, MAURICE R, TOPEKA
CASTEEL, CHARLES K, KC
CATHEY, ROBERT H, MANHATTAN
CAUBLE, WILBUR G, WICHITA
CAUGHRON, MICHAEL ROBERT, KC
CAVANAUGH, CLAIR J, GREAT BEND
CAVANAUGH, JOHN W, TOPEKA
CAVITT, ROBERT F, SHAWNEE MISSION
CAWLEY, LEO P, WICHITA
CECIL III, JOHN, HAYS
CENAC, MARK T, LEAVENWORTH
CHAFFEE, DEAN C, ABILENE
CHALIAN, ALEXANDER R, KC
CHAMBERLIN JR, CECIL R, TOPEKA
CHANEY, ERNIE J, BELLEVILLE
CHANG, CHAE H, KC
CHANG, FREDERIC C, WICHITA
CHANG, SHU FANG, SHAWNEE MISSION
CHANGPONG, SALA, LEAVENWORTH
CHAPMAN, JAMES H, WICHITA
CHAPPUIE, WILLIAM G, INDEPENDENCE
CHARD, FREDERICK H, WICHITA
CHAVALITHAMRONG, HANSA, HAYS
CHEDIAK, ELIAS, LAWRENCE
CHEN, JOHN S, HUTCHINSON
CHEN, TAK-MING, TOPEKA
CHENOWETH, JOHN R, D.D., OLATHE
CHERRY JR, ARTHUR C, TOPEKA
CHERVEN, PHILIP L, HUTCHINSON
CHEUNG, P W H, TOPEKA
CHIN, TOM D, KC
CHIU, CHU-SHIN, WICHITA
CHO, CHENG T, KC
*CHO, SECHIN, WICHITA
CHONKO, ARNOLD M, KC
CHOTIMONGKOL, ANUPONG, DODGE CITY
CHOUDHURY, M HASAN, COLDWATER
CHOW, STANLEY Y, FORT SCOTT
CHOY, JAMES K L, TOPEKA
CHRISTENSEN, MARION D, KIOWA
CHRISTIAN, STANLEY J, KC
*CHRISTMAN, CARL G, WICHITA
CHRONISTER, BERT, NEODESHA
CHUBB, RICHARD M, BAXTER SPRINGS
CHUN, CHUNG S, KC
CHUNG, JOHN J, SHARON SPRINGS
CISKEY, WILLIAM J, EUREKA
CLAASSEN, MILTON A, NEWTON
CLAIBORNE, RICHARD H, BAXTER SPRINGS
CLARK, COURTNEY, WICHITA
CLARK, CRAIG N, TOPEKA
CLARK, DAVID H, WAKEENEY
CLARK, LAURENCE A, WAMEGO
CLARK, LOELLA E, NEWTON
CLARK, MARGARET G, LAWRENCE
CLARK, ORVILLE R, ST PETERSBURG,FL
CLARK, RAY A, LAWRENCE
*CLEAVER, EDGAR M, WICHITA
CLIFTON, H DAVID, WICHITA
CLINTON, DALE L, LAWRENCE
CLYDE, HARRIE R, WICHITA
COALE, LLOYD H, KC
COBB, LESLIE H, MULVANE
COCHRAN, PAUL W, TOPEKA
CODY, DOROTHY, HAYS
CODY, JOHN, HAYS
COE, RICHARD O, SHAWNEE MISSION
COFFEY, ROY B, SALINA
*COHEN, JUSTIN T, WICHITA
COHEN, LOUIS, TOPEKA
COHEN, ROBERT A, SHAWNEE MISSION
COHEN, SIDNEY, TOPEKA
COHLMIA, JERRY B, WICHITA
COHN, STEVEN G, SHAWNEE MISSION
COHNBERG, ROSELLEN E, CEDAR VALE
COKELEY, JOHN M, TOPEKA
COLOSMITH, DONALD C, EMPORIA
COLE, WARD M, WELLINGTON
COLEMAN, GARY W, ABILENE
COLEMAN, THOMAS J, WICHITA
COLIP, F MERLYNN, NORTON
COLLIER, HAROLD W, WICHITA
COLLIER, WILLIAM J, MCPHERSON
COLLINS, DEAN T, TOPEKA
COLLINS, FRANCIS T, TOPEKA
COMBS, G RALPH, LEAVENWORTH
COMBS, PETER S, LEAVENWORTH
CONARD, CLAIR C, DODGE CITY
CONARD, RICHARD F, EMPORIA
CONCEPCION JR, EUGENIO S, WICHITA
CONNELLY, JOHN C, TOPEKA
CONRADY, PETER A, WICHITA
CONROY, ROBERT W, TOPEKA
COOK, BRUCE A, KC
COOK, O RAY, WICHITA
COOK, G EDWARD, WICHITA
COOK, JAMES O, KC
COOKE, ALLAN R, KC
COOMER, TYLER E, PITTSBURG
COOPER, ARTHUR E, NORTON
COOPER, JACK R, SHAWNEE MISSION
COOPER, KENT J, PITTSBURG
COOPER, LEO F, DREXEL,MO
COPELAND, GARY A, JUNCTION CITY
COPENING, TELL B, IOLA
CORBIN, MURRAY O, SHAWNEE MISSION
CORDELL, LARRY D, SHAWNEE MISSION
CORDER, ROBERT L, HIGHLAND
CORRIGAN, GEORGE F, WICHITA
COSSMAN, F PRICE, WICHITA
COTTON, ROBERT T, TOPEKA
COUCH JR, JAMES R, KC
COULTER, HENRY F, SHAWNEE MISSION
*COULTER, THAYNE A, CONCORDIA
COULTER, THOMAS B, KC
COVERT, THOMAS J, SALINA
COWLES, GEORGE E, WICHITA
COWLES, GORDON T, WICHITA
COX, ROBERT H, HAYS
COK III, IRA L, KC
COX JR, IRA, SHAWNEE MISSION
*COK, JACK A, .
COK JR, WALLACE F, KC
COYLE, JOHN F, COFFEYVILLE
CRAIG, CHARLES C, NEWTON
CRAM, ERNEST R, ST FRANCIS
CRAM JR, OLE R, LARNED
CRAMER, GUY W, PARSONS
CRAMM, RUSSELL E, HAYS
CRANE, C HERBERT, MANHATTAN
*CRANE, DAVID D, WICHITA
CRANSTON, STEPHEN D, NEWTON
CRARY, JOHN E, TOPEKA
CRAWFORD, ROBERT A, HUTCHINSON
CRIST, ROBERT O, KC
*CRISWELL, MILDRED LOUISE, WICHITA
CROCKETT, CHARLES A, KC
CRONEMEYER, RICHARD L, KC,MO
CRONIN, DONALD J, WICHITA
CROUCH, STEVEN W, KC,MO
CROUCH, WILLIAM H, TOPEKA
CROW, ERNEST W, WICHITA
CROWLEY, EDWARD K, WICHITA
CULP, LOUIS M, KC
CULTRON, FRANK T, SALINA
CULVER, WARREN T, LAWRENCE
CUMMINGS, RICHARD J, WICHITA
CURRAN, KEVIN E, SHAWNEE MISSION

D

DAEHNKE, SIGURD S, WINFIELD
DAHL, ASHER W, COLBY
DANIELS, ROBERT M, VALLEY CENTER
DARR, RICHARD B, SHAWNEE MISSION
DAUGHERTY, ROBERT M, MEADE
DAVENPORT, S SCOTT, WICHITA
DAVIDSON, HARRY T, WICHITA
DAVIS, CHESTER R, KC
DAVIS, CHRISTOPHER G, KC
DAVIS, DAVID M, LARNED
DAVIS, DAVID R, EMPORIA
DAVIS, GEORGE R, ELLSWORTH
DAVIS, PAUL M, WICHITA
DAVIS, RICHARD E, SHAWNEE MISSION
DAVIS, RONALD B, WICHITA
DAVIS JR, JAMES W, KC
DAY, HUGHES W, KC
DE BAKKER, JAN B, WICHITA
DE MOTT, WAYNE R, KC
DEAN, DAVID B, TOPEKA
DEBRIERE, SIDNEY L, PARSONS
DECHAIRO, THOMAS, WESTMORELAND
DECKER, DONALD D, HALSTEAD
DECKERT, ROSALIE E, WICHITA
DEGNER, JAMES B, GREAT BEND
DEITZ, MICHAEL R, KC
DEJONG, DAVID C, WICHITA
DELGADO, SERGIO, TOPEKA
DELLETT, KENNETH B, EL DORADO
DELP, MAHLON H, SHAWNEE MISSION
DELPHIA, ROBERT E, OLATHE
*DEMOSS, ELEANOR P, WICHITA
DENISON, TERRY R, SHAWNEE MISSION
DEPENBUSCH, FRANCIS L, HUTCHINSON
DEPOE, JOSEPH H, WINFIELD
DERRINGTON, KENNETH L, SHAWNEE MISSION
DETAR, GEORGE F, IOLA
DIACON, JAMES L, WELLINGTON
DIALLO, GASTON I, KC
DICK, ARTHUR R, KC
DICK, WILLIS G, IOLA
DICK JR, HENRY J, CHANUTE
DICKERSON, SHELBY C, KC
DICKINSON, CHARLES R, COFFEYVILLE
DIEDERICH, DENNIS A, KC
DIEHL, ANTONIO M, SHAWNEE MISSION
DIENER, CLAYTON H, HESSTON
DIVER, ROBERT W, GULFPORT,MS
DLABAL, FRANK A, WILSON
DOBRATZ, ROBERT A, BELOIT
DOCKHORN, ROBERT J, SHAWNEE MISSION
DOHERTY, WILLIAM R, SHAWNEE MISSION
DONAHOE, DORVAL H, MEADE
DONNELL, JAMES M, WICHITA
DONNELLY, FRANCIS M, SHAWNEE MISSION
DOORNBOS, J FRED, WICHITA
DOUBEK, HERBERT O, BELLEVILLE
DOUGHERTY, THOMAS M, GARNETT
DOUTHIT, JOHN D, MANHATTAN
DOWELL, JAMES C, SALINA
DOZIER, FRED S, HERINGTON
DRAEMEL, H RICHARD, SALINA
DRAKE, DOUGLAS J, BELOIT
DRAKE, RALPH L, WICHITA
DREHER, HENRY S, SALINA
DREVETS, CURTIS C, WICHITA
DRIGGS, GUY K, WICHITA
DUCKETT, THOMAS G, HIAWATHA
DUCKETT II, THOMAS G, SHAWNEE MISSION
DUDDING, BURTON A, KC
*DUICK, GREGORY, WICHITA
DUJOVNE, CARLOS A, KC

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DUNAGIN, JACK A, TOPEKA
DUNLAP, RICHARD L, LAWRENCE
DUNN, DANIEL R, COLBY
DUNN, MARVIN I, KC
DUNSHEE, CARLYLE M, FORT SCOTT
DURANO, ANTONIO C, WICHITA
DURKEE, WILLIAM R, MANHATTAN
DURST JR, ROBERT D, TOPEKA
DUYSAK, SAMI, LEAVENWORTH
OWORZAK, DAVID L, WICHITA
DYCK, ARTHUR H, MCPHERSON
DYCK, CORA E, WICHITA
DYCK, GEORGE, WICHITA
DYER, VERNON E, WICHITA

E

EATON, GLEN E, SALINA
EATON, LESLIE F, SALINA
ECKART, DE MERLE E, HUTCHINSON
ECKERT, WILLIAM G, WICHITA
EDDY, MURRAY C, HAYS
EDDY, VICTOR M, HAYS
EDROZO, M LUZ, COFFEYVILLE
EDWARDS, DAVID J, EMPORIA
EDWARDS, MANIS C, WICHITA
EGEA, FERNANDO M, KC
EGELHOF, RICHARD H, WICHITA
EICHHORN, FRANK D, GARDEN CITY
EIDT, DAVID W, OLATHE
EIDT, LAURENCE A, OLATHE
EIKERMANN, WILLIAM C, SHAWNEE MISSION
ELDER, DOUGLAS M, TOPEKA
ELLEDGE, E FRED, GREAT BEND
ELLIS, HARVEY D, WICHITA
ELLIS, STEPHEN S, COFFEYVILLE
ELLISON, PAUL D, SALINA
ELNEN, WALTER T, WICHITA
*ELNESER, RAFAEL, WICHITA
EMERY, FRANK A, WICHITA
EMMOT, WILLIAM W, INDEPENDENCE
EMPSON, CHARLES L, INDEPENDENCE
ENBERG, ROBERT, HAYS
ENDERS, WRAY, SHAWNEE MISSION
ENNS, EUGENE K, NEWTON
ENNS, JAMES H, NEWTON
ENS, GERHARD GEORGE, HILLSBORO
ENS, PETER, HILLSBORO
ENSEY, T CRANFORD, MARION
ERICKSON, CLARENCE W, PITTSBURG
ERKEN, RONALD V, WICHITA
ESCH, JOHN G, PITTSBURG
ESPEDIDO-RAMIRZ, IRENE P, PITTSBURG
ESRIG, HAROLD L, D.O., SHAWNEE MISSION
ETZENHOUSER III, RUSSELL D, SHAWNEE MISSION
EVANS, FARRIS D, WICHITA
EVANS, GRANT E, WICHITA
EVANS, KENNETH L, WICHITA
EVANS, WILLIAM R, GREAT BEND
EVANS JR, WILLIAM E, SHAWNEE MISSION
EWING, THOMAS D, LARNED

F

FAIRCHILD, JOHN A, MANHATTAN
FALTER, RICHARD T, HUTCHINSON
FARHA, GEORGE J, WICHITA
FARHA, S JIM, WICHITA
FARRAR JR, WILLIAM H, ARKANSAS CITY
FAST, ROBERT E, ATCHISON
FAST, W SPENCER, ATCHISON
FEAGAN, JERRY, TOPEKA
FEIGHNY, ROBERT E, SALINA
FELT, SAMUEL E, COLUMBUS,GA
FENDER JR, THOMAS H, WICHITA
FENT, LEE S, NEWTON
FERNANDEZ, LUIS A, TOPEKA
FERNIE, ROBERT W, HUTCHINSON
FESTOFF, BARRY W, KC
FERRIS, BRUCE G, WICHITA
FEUILLE, JR, EDMOND G, WICHITA
FIELD, RICHARD A, TOPEKA
FIELDMAN, E JAY, SAN DIEGO,CA
FIELDS, GALEN W, SCOTT CITY
FIESER, CARL W, GREAT BEND
FILLEY, VERNON W, PRATT
FILLMAN, ELDON M, TOPEKA
FINK, ABRAHAM A, PLANTATION,FL
FINLEY, DENNIS R, WICHITA
FIRKINS, RICHARD T, WICHITA
FISCHER, REX R, MANHATTAN
FISHER, JAMES B, WICHITA
FITZGERALD, EDWARD J, WICHITA
FITZPATRICK, M ROBERT, KC
FIXLEY, MARK S, SHAWNEE MISSION
FLANDERS, H ALDEN, HAYS
FLEMING, FORNEY W, WICHITA
FLECKENSTEIN, CHARLES S, ONAGA
FLOERSCH, HUBERT M, KC
FLOWERS JR, CLELL B, WICHITA
FORD, CHARLES R, WICHITA
FORD, FRED L, TOPEKA

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FORDYCE, NORMAN, KC
FORET, JOHN O, KC
FORSTER JR, LOUIS G, SALINA
FORTUNE, CEDRIC B, OLATHE
FOSS, DANIEL C, HUTCHINSON
FOSTER, CHARLES G, TOPEKA
FOSTER, D BERNARD, TOPEKA
FOSTER, FRANCES M, KC
FOWLER, ROBERT J, WICHITA
FOWLER, WAYNE L, CONCORDIA
FOX, DEANNA K, KC
FOX, HOWARD A, KC
FRANCIS, NORTON L, WICHITA
FRANCISCO, CLARENCE L, KC
FRANCISCO, W DAVID, KC
FRANSEN, HERBERT, NEWTON
FRANSEN, PAUL H, NEWTON
FRANZ, ROBERT G, HILLSBORO
FRAZIER, RICHARD L, EMPORIA
FREDERICK, MAC F, HUGOTON
FREDRICKSON, DUANE E, LINDSBORG
FREEBORN JR, WARREN S, CLYDE
FREEMAN, F GILES, PRATT
FREEMAN, FRED A, MANHATTAN
FREEMAN, RAYMOND S, SALINA
FRIESEN, DALE, LAWRENCE
FRIESEN, FLORENCE V, HESSTON
FRIESEN, ORLANDO J, BUHLER
FRIESEN, STANLEY R, KC
FRIGGERI, ROBERT W, GIRARD
FRITZ, GEORGE E, WICHITA
FRITZEMEIER, WILLIAM H, WICHITA
FROMM, ARTHUR H, WICHITA
FRY, LUTHER L, GARDEN CITY
FULLEN, JERYL G, SHAWNEE MISSION
FULLER, DERYL D, LINDSBORG
FULTON, JOHN K, WICHITA
FUNES, CARLOS A, EMPORIA
FUNK, EDWARD D, KC

G

GABATIN, ANGELITA S, SAN FRANCISCO,CA
GABATIN, JOSE Q, SAN FRANCISCO,CA
GAINES, LARRY S, WICHITA
GALICHIA, JOSEPH P, WICHITA
GALLEHUGH, KEITH W, KC
GALVAN, ALONSO, WICHITA
GANN, E LAMONTE, EMPORIA
GANS, FREDERICK A, SALINA
GANZARAIN, RAMON C, TOPEKA
GARCIA, GOULD C, EMPORIA
GARDNER, GLENN M, SHAWNEE MISSION
GARDNER, JAMES D, MANHATTAN
GAREY, WILLIAM JOHN, INDEPENDENCE
*GARWOOD, JAN, LYONS
GATEND, JOSEPH, GREAT BEND
GAY, JOHN D, TOPEKA
GEHRT, EARL B, CHANUTE
GEITZ, JAMES M, EMPORIA
GELVIN, E RAYMOND, CONCORDIA
GENCH, RAYMOND L, CARMEL,CA
GENOEL, EVALYN S, SAN FRANCISCO,CA
GENOEL, JOSEPH E, TOPEKA
*GENILD, AMANCIO C, WICHITA
*GENILD, CELESTE A, WICHITA
GENTRY, KALE C, SHAWNEE MISSION
GEORGE, EARL F, WICHITA
GEORGE, M DON, WICHITA
GERBER, HARRY A, LEAVENWORTH
GERJARUSAK, PRAPAS, LEAVENWORTH
GESSLER, DONALD J, WICHITA
GETTLER, DEAN T, FORT SCOTT
GIBBS, EUGENE, COFFEYVILLE
GILBERT, J HOWARD, SENECA
GILBERT, ROBERTA M, SHAWNEE MISSION
GILBERT II, JOHN H, GARDEN CITY
GILHOUSEN, FREDERIC M, KC
*GILMARTIN, RICHARD C, WICHITA
GILL, GEORGE L, LAMPE,MO
GILLEN, BILLY A, SHAWNEE MISSION
GILLES, HELEN M, LAWRENCE
GILMORE, CLARENCE A, KC
GINAVAN, DUANE A, EMPORIA
GIROD, CHARLES I, EL DORADO
GIVNER, DAVID, WICHITA
GLAZZARD, CHARLES D, OLATHE
GLEASON, JIMMIE A, TOPEKA
GLENN, JAMES N, EMPORIA
GLENN, LYLE G, PROTECTION
GLOVER, RICHARD M, NEWTON
GNAU, FREDERIC B, HALSTEAD
GODFREY, ROBERT G, KC
GODFREY, WILLIAM A, KC
GODWIN, PHILLIP A, LAWRENCE
GOERING, DONALD D, SALINA
GOERING, EMIL L, TOPEKA
GOERING, ROBERT C, WICHITA
GOERTZ, LED R, KC
*GOLD, JAY H, WICHITA
GOLDBERG, HERBERT R, WICHITA
GOLDSTEIN III, NATHAN, KC
GOLLERKERI, MOHAN P, SHAWNEE MISSION

GOLLIER, ROBERT A, OTTAWA
GOLLIER II, ROBERT A, OTTAWA
GOMEZ, FRANCISCO, SHAWNEE MISSION
GOMEZ, MODESTO S, PITTSBURG
GONZALEZ, FRANCISCO, WICHITA
GONZALEZ, HIRAM, WICHITA
GOOD, JAMES T, FORT SCOTT
GOOD, WENDELL LISLE, SHAWNEE MISSION
GOOGIN, ETHELYNN A, WICHITA
GOODIN, WALKER D, WICHITA
GOODPASTURE, HEWITT C, WICHITA
GOODPASTURE, WILLARD C, HUTCHINSON
GOODWIN, DONALD W, KC
GOOTEE, JOSEPH E, TOPEKA
GOTO, HIROSHI, KC
GOULDNER, RENE M, WICHITA
GOYLE, KRISHAN K, WICHITA
GOYLE, VIMAL, WICHITA
GRADINGER, BILLENS C, HALSTEAD
GRADY, KENNETH L, SHAWNEE MISSION
GRAHAM, J MALCOLM, LEAVENWORTH
GRAHAM, KENNETH L, LEAVENWORTH
GRAHAM, THOMAS W, LEAVENWORTH
GRAHAM JR, CHARLES P, TOPEKA
GRANTHAM, JARED J, KC
GRAUEL, CHARLES W, WICHITA
GRAUEL, TRUMAN W, WICHITA
GRAVES, JACK W, WICHITA
*GRAY, C LUCIEN, WICHITA
GRAY, CAPTAIN K, LAWRENCE
GRAY, DAVID E, TOPEKA
GRAY, H TOM, WICHITA
GRAYIB, ANTOINE S, TOPEKA
GRAYSON, ROY D, ALBUQUERQUE,NM
GREEN, LAWRENCE C, ARKANSAS CITY
GREENBERG, GEORGE E, GARDEN CITY
GREENBERG, MARK, TOPEKA
GREENBERG, MARTIN TAYLOR, KANSAS CITY,MO
GREENBERGER, N J, KC
GREENE, HORACE T, TOPEKA
GREENWOOD, EDWARD D, TOPEKA
*GREER, JAMES A, WICHITA
GREER, RICHARD H, TOPEKA
GREGORY, ARLENE, PITTSBURG
GRIBBLE, ROBERT N, WICHITA
GRIFFING, RICHARD B, SALINA
GRIGSBY, KENNETH R, SUN CITY,AZ
GRIGSBY JR, GEORGE T, TOPEKA
GRILLOT, FLOYD B, WICHITA
GRIMES, I ROSS, LIBERAL
GRIMES, JAMES T, LYONS
GRIPKEY, CLARENCE A, KC
GRISOLIA, ANDRES, LEAVENWORTH
GRISWOLD, DALE G, NEWTON
GROHS, HEINZ K, WICHITA
GROVE, JOHN A, NEWTON
GROWNEY, JOHN T, ATCHISON
GRUENDEL, RICHARD A, KC
GRUENDEL, VIRGINIA T, KC
GRUND, FRANK M, WICHITA
GRUSHNYS, ARNOLD, WICHITA
GSELL, GEORGE F, WICHITA
GUERRA, TOMAS H, MARION
GUMUCIO, MARIO L, KC
GUNN, MARVIN R, SALINA
GUNTER, CARL C, DUINTER
GURWITH, MARC, KANSAS CITY
GUTHRIE, RICHARD A, WICHITA

H

HA, SANG W, COFFEYVILLE
HAAS, CHARLES O, KC
HABERSANG, ROLF W D, KC
HADLEY, DELMONT C, OTTAWA
HAFFNER, WILLIAM N, EL DORADO
HAGAN, C THOMAS, WICHITA
HAGAN, FRANCIS J, WICHITA
HAIGLER, JAMES P, HAYS
HAINES, CHESTER W, HAVEN
HAINES, ROBERT A, TOPEKA
HALE, RALPH, WICHITA
HALL, JERRY D, ANTHONY
HALL, WESLEY H, GIRARD
HALLEY, M MARTIN, TOPEKA
HALLING, L WILLIAM, HAYS
HALPIN, EDWARD D, WICHITA
HALVORSON, HOWARD C, OLATHE
HAMILTON, JAMES J, WAKEENEY
HAMM, ORVAL L, SIALKOT,
HAMMEKE, JOHN C, LEAVENWORTH
HAMMEL, GEORGE W, EL DORADO
HAMTIL, LAWRENCE W, SHAWNEE MISSION
HAN, CHAN S, COFFEYVILLE
HANCOCK, ALAN C, KC
HANSON, DAVID C, IOLA
HARA, GLENN S, KC
HARD, BENJAMIN F, SHAWNEE MISSION
HARDIN, CREIGHTON A, KC
HARMS, ALBERT C, SHAWNEE MISSION
HARMS, EDWIN M, WICHITA
HARMS, WILMER A, HALSTEAD
HARNED, BERT W, SHAWNEE MISSION

HARPSTER, GENE O, SHAWNEE MISSION
HARRIS, FRANK H, WICHITA
HARRIS, HUBERT L, TOPEKA
HARRIS, NORMAN R, SALINA
HARRIS, NORVAN O, LIBERAL
HARRIS, PATRICIA A, TOPEKA
HARRIS JR, CLAIB B, GARNETT
HARRISON, A BROOKS, WICHITA
HARRISON, HALL E, TOPEKA
HART, OILLIS L, WICHITA
HART, LAWRENCE E, ATCHISON
HARTENBOWER, CAREY A, SALINA
HARTLEY, FOUNT K, HONOLULU,HI
HARTLEY, ROY W, NORTON
HARTMAN, CHARLES R, KC
HARTMAN, GERALD Y, KC
HARTMAN, ROGER L, NORTON
HARTOCOLLIS, PETER, TOPEKA
HARVEY, JOHN E, EMPORIA
HARVEY, ROSEMARY B, WICHITA
HARVEY, THOMAS S, WICHITA
HARWOOD, CLAUDE J, GLASCO
HASKINS, ROBERT J, CHANUTE
HASSELLE III, JAMES E, TOPEKA
HASSETT, GERARD R, COLBY
HASTINGS, MARY T, SHAWNEE MISSION
HATHAWAY, PETER, KC
HATTON, DONALD W, LAWRENCE
HATTON, LLOYD W, SALINA
HATTRUP, RICHARD J, WICHITA
HAYENHILL II, MARSHALL A, EMPORIA
HAWKEY, ALFRED S, NEWTON
HAWLEY, RAYMOND G, WICHITA
HAYDEN, ABIGAIL, FREDONIA
HAYES, WILLIAM L, WICHITA
*HAYS, THOMAS H, WICHITA
HEARD, KENNETH M, GARDEN CITY
HEASTY, ROBERT G, MANHATTAN
HEBBAR, SATYA N, TOPEKA
HEDRICK, KENNETH E, HUTCHINSON
HEINS, LAWRENCE G, ABILENE
HENDERSON, CHARLES F, PARSONS
HENDERSON, JOHN M, KC,MO
HENNEBERGER, CHARLES E, ATWOOD
HENNING, CALVIN W, OTTAWA
HENNING, CHARLES E, WICHITA
HENSLEY JR, CLINE O, WICHITA
HEREO, JOHN, WICHITA
HERMES, RICHARD L, LAWRENCE
HERMRECK, ARLO S, KC
HERNANDEZ, ALEXANDER M, GARDEN CITY
HERRERA, JORGE J, TOPEKA
HERSHORN, SIMON E, WICHITA
HERZON, CHARLES O, SHAWNEE MISSION
HESSE, ROBERT S, WELLINGTON
HESSER, HERBERT H, SHAWNEE MISSION
HIEBERT, ABRAHAM E, WICHITA
HIEBERT, DAVID L, LAWRENCE
HIEBERT, JOHN B, TOPEKA
HIEBERT, PETER E, KC
HIESTERMAN, HERMAN W, QUINTER
HIGGINS, RONALD A, SHAWNEE MISSION
HILOYARD, VICTOR H, COLBY
HILOYARD II, VICTOR H, COLBY
HILL, JAMES E, ARKANSAS CITY
HILL, JOHN J, HUTCHINSON
HILL, PHILLIP K, NEWTON
HILL, RICHARD H, MEADE
HILL, ROBERT N, TOPEKA
HINSHAW, ALFRED H, WICHITA
HINSHAW, CHARLES T, WICHITA
HINSHAW, EDGAR O, ARKANSAS CITY
HINSHAW JR, CHARLES T, HUTCHINSON
HINTHORN, DANIEL R, KC
HIRATZKA, TOMIHARU, WICHITA
HIRD, WAYNE E, LAWRENCE
HIRSCH, MARTIN S, KC
HIRSCHBERG, J COTTER, TOPEKA
HITCHCOCK, CHAS THOMAS, KC
HOBBS, DONALD D, TOPEKA
HOBSON, MILBURN W, SHAWNEE MISSION
HODES, HERBERT C, SHAWNEE MISSION
HODGES, BRUCE E, SHAWNEE MISSION
HODGES, GLENN R, KC
HODGES, MERLE A, SALINA
*HODGSON, ROBERT B, WICHITA
HODSON, HERVEY R, WICHITA
HOFER, DEWAYNE D, CONCORDIA
HOFFER, JOHN G, MEDICINE LODGE
HOHERZ, DAVID G, TOPEKA
HOLCOMB, DONALD G, LIBERAL
HOLCOMB, WILLIAM M, LIBERAL
HOLDCRAFT, JACQUELYNE, KC
HOLDEN JR, RAYMOND F, WICHITA
HOLDERMAN, WALLACE D, HUTCHINSON
HOLLADAY, HARMON H, WICHITA
HOLLADAY, KENNETH R, EUDORA
HOLLEMAN JR, JAMES F, D.O., KC
HOLLOWELL JR, JOSEPH G, KC
HOLMAN, JON B, SALINA
HOLMES, FREDERICK F, KC
HOLMES, GRACE E, KC
HOLSINGER, DONALD M, PITTSBURG
HOLT, JOHN M, GREAT BEND
HOLT, ROBERT G, HALSTEAD
HOOFER, WILFORD D, HALSTEAD
HOOGSTRATEN, BARTH, KC
HOPKINS, WILLIAM O, SHAWNEE MISSION
HOPKINS JR, B MORRISON, SCOTT CITY
HOPKINS JR, LENLY T, SHAWNEE MISSION
HOPPER, CHARLES R, EMPORIA
HORBELT, DOUGLAS V, WICHITA
HORNE, JAMES B, TOPEKA
HORSEMAN, ROBERT F, SHAWNEE MISSION
HORTON, WILLIAM A, KC
HOSTETTER, PHILIP H, MANHATTAN
* HOUSHOLDER, DANIEL F, WICHITA
*HOUSHOLDER, MARTHA S, WICHITA
HOWARD, DONALD O, WICHITA
HOWARD II, W ROBERT, ARKANSAS CITY
HOWERTER JR, BERNARD E, COFFEYVILLE
HOYLE, WINTHROP E, WINFIELD
HOYT, ARTHUR W, TOPEKA
HSU, CHENG H, TOPEKA
HUAMAN, ANTONIO M, TOPEKA
HUDSON, JAMES R, CHANUTE
HUDSON, ROBERT P, OLATHE
HUEBERT, DEAN A, WICHITA
HUERTER, DAVID F, PITTSBURG
HUERTER, QUENTIN C, KC
HUFF, JOHN D, KC
HUFFMAN, DAVID H, KC
HUGHES, JOHN JAY, KANSAS CITY
HUGHES, ROBERT WALTER, LAWRENCE
HULL, KENNETH L, WICHITA
HULL, NORMAN E, HAYS
HULTGREN, MYRON K, WICHITA
HUME, JOSEPH W, WICHITA
HUMMER, LLOYD M, WICHITA
HUMPHREY, LOREN J, KC
HUNTER JR, JAMES S, MANHATTAN
HUNTER, KENNETH R, LEBO
HURST, THOMAS C, WICHITA
HURWITZ, ARYEH, KC
HUSTEAD, ROBERT F, WICHITA
HUSTON, FRANCIS W, WINCHESTER
HUSTON, JOSEPH W, TOPEKA
HUTCHISON, GLEN C, HAYS
HUTTON, FREDERICK A, TOPEKA
HUYCKE DUDGEON, M MAUREEN, KC
HWA, EUGENE C, NEWTON
HYDE, MARSHALL E, FT SMITH,AR
HYLWA, THEODORE M, SHAWNEE MISSION
HYNOMAN, H HAROLD, WICHITA
HYNES, HENRY E, WICHITA

I

IBARRA, J LUIS, WICHITA
IBARRA, RICHARD C, KC
INGHAM JR, H LAIRD, LAWRENCE
INGRAM, JOHN E, KC
INNES, ROBERT C, KC
IRBY, ADDISON C, FORT SCOTT
IRBY, PRATT, FORT SCOTT
ISAAC, CHARLES A, NEWTON
ITURRALDE, GEORGE, SHAWNEE MISSION

J

JACKMAN, GLENN H, ESTES PARK,CO
JACKS, J WARREN, PRATT
JACKSON, CHARLES R, WICHITA
JACKSON, LINDA H, TOPEKA
JACKSON, ROGER P, KC
JACKSON, VICTOR L, ALTAMONT
JACKSON JR, OELMAS A, SALINA
JACOB, KANNAMPALLY L, EL DORADO
JACOBS, DAVID S, KC
JACOBS, RAE R, KC
JACOBY II, ROBERT E, TOPEKA
JAHANIAN, DARYOUSH, KC
JAKOWATZ, JAMES G, KC
JAMBOR, JAMES J, DODGE CITY
JAMES, VERNON L, WICHITA
JANES, DONALD R, SHAWNEE MISSION
JANSSEN, ERWIN T, TOPEKA
JANZEN, HERMAN F, HILLSBORO
JARROTT, JOHN B, HUTCHINSON
*JAWADI, M HUSAIN, WICHITA
JAYARAM, MARANDAPALLI R, SHAWNEE MISSION
JAZAYERLI, NABIL, WICHITA
JEHAN, SAYED S, WICHITA
JENKINS, EDWARD, GARDEN CITY
JENKINS, NEAL M, SALINA
JENNEY, CHARLES B, WICHITA
JENSEN, THOMAS M, OLATHE
*JESTER, SHELBY L, WICHITA
JEWELL, ROSS L, ST FRANCIS
JEWELL, WILLIAM R, KC
JOHLER, TERRY H, SHAWNEE MISSION
JOHNS JR, LEO F, KC
JOHNSON, FREDERICK F, TOPEKA
JOHNSON, HOWELL D, DODGE CITY
JOHNSON, J RICHARD, MCPHERSON
JOHNSON, JOHN E, KC
JOHNSON, JOSEPH H, EL DORADO
JOHNSON, PAUL D, LEAVENWORTH
JOHNSON, THOMAS E, WICHITA
JONES, CHARLES E, SHAWNEE MISSION
JONES, EDWARD L, GREAT BEND
JONES, FORREST H, COLUMBUS
JONES, HIVOR, SHAWNEE MISSION
JONES, H PENFIELD, LAWRENCE
JONES JR, HERMAN H, KC
*JORDON, DAVID W, WICHITA
JORG, HELEN H, SHAWNEE MISSION
JOSEPH, BRIAN W, TOPEKA
JOSEPH, HOWARD F, LAWRENCE
JOSS, CHARLES S, TOPEKA
JOUVENAT, NEIL C, SHAWNEE MISSION
JOYCE, G BERNARD, TOPEKA
JUBELT, HILBERT P, MANHATTAN
JUDILLA, FRANCISCO JR, WICHITA
JUMPER, JERRY L, D.O., SABETHA
*JUNOD, FORREST LEE, WICHITA
JUSTUS, WILLIAM J, PLEASANTON

K

KADISON, HERBERT I, WICHITA
KAELSON JR, PAUL A, WICHITA
KAGAN, STUART M, SHAWNEE MISSION
KALBAC, RICHARD W, GARDEN CITY
KALDOR, RICHARD H, MANHATTAN
KALIVAS, JAMES T, KC
KANE JR, WILLIAM M, HAYS
KAROATZKE, E STANLEY, WICHITA
KARDATZKE, JON K, WICHITA
KASHA, ROBERT L, WICHITA
KASSEBAUM, GLEN E, EL DORADO
*KASSEBAUM, KENNETH G, WICHITA
KASSEL, HENRY W, LAKEWOOD,CO
KATZ, JEROME B, TOPEKA
KAUFFMAN, RALPH E, KC
KAUFMAN, EUGENE E, WICHITA
KAUFMAN, LELAND R, WINFIELD
KAUFMAN, WILLARD E, MOUNDRIDGE
KAUL, ANAND N, WINFIELD
KAYEL, KARL K, TOPEKA
KEENE, GEORGE H, WICHITA
KEENY, M GARY, WICHITA
*KELLER, JAMES P, WICHITA
KELLY, CHARLES R, KC,MO
KELLY, DAN A, TOPEKA
*KELLY, ROBERT W, WICHITA
KEMPTHORNE, CHARLES R, MANHATTAN
KENDALL, TOM E, WICHITA
KENDRICK, J GILLERAN, WICHITA
KENNEDY, GERALD T, WICHITA
KENNEDY, HOWARD U, TOPEKA
KENNEDY, JAMES A, KC
KENNEDY, KENNETH R, SHAWNEE MISSION
KEPES, JOHN J, KC
KERBY, GERALD R, KC
KESTENBAUM, THELDA M, KC
KETCHUM, LYNN D, KC
KEYS JR, ROBERT C, TOPEKA
KEYS SR, ROBERT C, WINCHESTER
KHICHA, GYANCHAND J, WICHITA
KHOURY, GEORGE H, WICHITA
KHURANA, SATISH K, SHAWNEE MISSION
KIFER, C JAMES, HAYS
KIHM, ALBERT A, CHANUTE
KIM, JONG M, KC
KIM, PAIK N, WICHITA
KIM, SOON EUNG, WICHITA
KIM, YONG W, TOPEKA
KIM, YUNG SUP, SAN FRANCISCO,CA
KIMBALL, RICHARD R, MANKATO
KIMBLE, JAMES A, WICHITA
KIMBROUGH, VICTORIA F, LAWRENCE
KIMURA, CHARLES C, SHAWNEE MISSION
KINDLING, PAUL H, TOPEKA
KING, CHARLES R, KC
KING, WILLIAM T, GREAT BEND
KINNAN, L F, CALDWELL
KIRBY, MERLIN G, GREAT BEND
KIRCHNER, FERNANDO R, KC
KIRK, THOMAS E, MANHATTAN
KIRK JR, E DAVID, WICHITA
KIRKEGAARD, RODGER S, TOPEKA
KIRSHNER, NANCY A, KC
KISER, JOHN L, WICHITA
KISER, WILLARD J, WICHITA
KITCHEN, ROBERT R, WICHITA
KLASSEN, DANIEL S, NEWTON
KLAUS, HANNA, WICHITA
KLEMMER, HERBERT, TOPEKA
KLENDA JR, MARTIN B, BELOIT
KLIEWER, VERNON L, NEWTON
KLINGLER JR, EUGENE A, MANHATTAN
KNAPP, LESLIE E, WICHITA
KNAPP, M ROBERT, WICHITA
KNAPPENBERGER, ROY C, WICHITA

* Probationary members.

KNECHT, STEPHEN M, EMPORIA
KNEIDEL, THOMAS W, WICHITA
KNUTH, KENNETH L, INDEPENDENCE
KODANAZ, A AYTEKIN, SHAWNEE MISSION
KOENEKE, IRENE A, HALSTEAD
KOONS, JESS W, LIBERAL
KOSAR, CLARENCE O, CONCORDIA
KOURI, SAMMY H, WICHITA
KOVARIK, ERNEST D, TOPEKA
KOZIKOWSKI, BEN M, SHAWNEE MISSION
KOZNEK OO, CLARE, OSKALOOSA
KRANTZ, KERMIT E, KC
KRAUSE, ROLAND L, WICHITA
KROLL, HARRY G, TOPEKA
KRUEGER, ALAN L, KC
KRUEGER, HAVEN C, GREAT BEND
KUBIN, DORIS A, SHAWNEE MISSION
KULLMAN, FREDERICK M, LOUISBURG
KUNNAWUTHIDEE, KAMOLTIPYA, DODGE CITY
KURTH, C JOSEPH, WICHITA
KURTH, ROBERT H, SHAWNEE MISSION
KUTILEK, FRANK J, WICHITA
KWEE, SIOE T, KC
KYNER, JOSEPH L, KC

L

LABHSETWAR, SNEHALATA A, JUNCTION CITY
LAFENE, BENJAMIN W, MANHATTAN
LAHAM, ALEXANDER J, WICHITA
*LAI, JENG Y, WICHITA
LAING, ROBERT R, KC
LAIRD, DALE D, OLATHE
LAKE, MAX S, SALINA
LAMBRIGHT, ROBERT L, SHAWNEE MISSION
LANCE, RAYMOND W, PITTSBURG
LANCE JR, JOHN F, WICHITA
LANSKY, LESTER L, KC
LANSKY, SHIRLEY B, KC
LAPI, RUTH M, SHAWNEE MISSION
LARSON, DELBERT L, HIAWATHA
LASLEY, MICHAEL B, HAYS
LASLEY, DAVID A, SALINA
*LATIMER, KATHERINE, WICHITA
*LAUVER, MARY ANN, WICHITA
LAURY, DAVID G, OTTAWA
LAVAVESHKUL, CHIRUNO, PARSONS
LAW, FINDLEY, ELLINWOOD
LAWLESS, HAROLD L, BLUE RAPIDS
LAWN, RAYMOND A, WICHITA
LAWS, LEWIS R, MARYSVILLE
LAWSON, DWIGHT, TOPEKA
LAWSON, ROBERT C, TOPEKA
LAWTON, MARVIN K, CONCORDIA
LAYBOURNE JR, PAUL C, KC
LAZAR, HARRY, WICHITA
LEAKE III, HUNTER C, KC
LEARNED, GEORGE R, LAWRENCE
LEATHERS III, HOLLIS K, SHAWNEE MISSION
LEE, JAE M, KC
LEE, KYO R, KC
LEE, R REX, WICHITA
*LEE O O, ROBERT PAUL, OSKALOOSA
LEE, SONG DOW, TOPEKA
LEE, SONG PING, TOPEKA
LEE JR, EDWARD S, WICHITA
LEE JR, JAMES G, KC
LEE, JOSEPH R, ASHLAND
LEFFLER, PAUL B, PITTSBURG
LEGASPI JR, PEDRO L, SHAWNEE MISSION
LEGER, LEE H, FT MYERS,FL
LEIGH, LAWRENCE E, SHAWNEE MISSION
LEIKER, JOSEPH, WICHITA
LEITE, CAROL JO, KC
LEISY, JERALD W, WICHITA
LEITCH, DAVID A, GARNETT
LEMOINE JR, ALBERT N, KC
LENEVE, ROBERT T, HUGOTON
LENSKI JR, FRANCIS X, IOLA
LENTZ, WILLIAM R, TOPEKA
LEO, WILLIAM A, KC
LESSENDEN, GLENN A, LAWRENCE
LESSENDEN JR, C M, TOPEKA
LETTNER, HANS T, HUTCHINSON
LEVINE, ERROL, KC
LEVY, EDWIN Z, TOPEKA
LEWIN, WALTER, SHAWNEE MISSION
LEWIS, JAMES E, SHAWNEE MISSION
LEWIS JR, H DANIEL, KC
LIES, BARTHEL N, COLWICH
LIES, JAMES E, WICHITA
LIN, JOE J, WICHITA
LIN, TUNG K, TORRANCE,CA
LINDLEY, MILTON E, WICHITA
LINDSLEY, CAROL B, KC
LINDSLEY, HERBERT B, KC
LINHARDT, RONALD O, WICHITA
LINSHAW, MICHAEL A, KC
LIPSCHITZ, DAVID A, KC
LIPSEY, JAMES H, SHAWNEE MISSION
LITTLE, L GILBERT, WICHITA
LIU, CHIEN, KANSAS CITY
LIUJULUSCHAN, RIT, PARSONS
LLOYD, HARVEY L, KC
LLOYD, JAMES W, CONCORDIA
LOCKHART, JOSEPH G, WICHITA
LOEFFLER, JAMES A, WICHITA
LOEWEN, HENRY H, WICHITA
LOEWEN, PETER S, SHAWNEE MISSION
LOEWEN, WILLIAM C, WICHITA
LOFGREEN, VICTOR J, OTTAWA
LOGAN, GEOFFREY G, WICHITA
LOGANBILL, VARDEN J, MOUNDRIDGE
LOHMEYER, KENNETH L, EMPORIA
LONEY, JOHN M, BELOIT
LONG, EDWARD E, HUMBOLDT
LONG, JOHN W, TOPEKA
LONG, LLOYD O, GOODLAND
LONG, ROBERT C, NORTON
LONGWOOD, ORLIN W, TUCSON,AZ
*LORCH, VICHIEN, WICHITA
LOTITO, CARLOS A, SHAWNEE MISSION
LOVETT, PAUL A, WICHITA
LOW, HAROLD L, WICHITA
LOW, JOSEPH H, SUN CITY,AZ
LOWE, DEWITT S, HIAWATHA
LOWE, STANLEY W, MANHATTAN
LOWMAN, JAMES T, KC
LOWRY, RAY F, EL DORADO
LUEDTKE, WALTER E, EMPORIA
LUEKEN, LUEKE B, WICHITA
LUELLEN, THOMAS J, WICHITA
LUKENS, DAVID, HUTCHINSON
LUKERT, BARBARA P, KC
LULO, ANTONIO R, SHAWNEE MISSION
LUMB, RAYMOND C, TOPEKA
LUNGSTRUM, JACK E, SALINA
LUZZATI, ENZO F, WICHITA
LYNCH, SEAN R, KC
LYNCH, JOHN A, TOPEKA
LYONS, DAVE J, PITTSBURG
LYONS JR, FRANK C, MANHATTAN

M

MACARTHUR, RICHARD IAN, KANSAS CITY
MAC KILLOP JR, DANIEL, WINFIELD
MACE, RONALD D, JUNCTION CITY
MACY, NORMAN E, SALINA
MACY, TED L, SALINA
MADDEN, WILLIAM J, LINCOLN
MADISON, WILLARD A, NORTONVILLE
MADISON JR, WARD N, WICHITA
MADSEN, GLENN L, LAWRENCE
*MAGIOSON, ELLIOT ARTHUR, WICHITA
MAKLAD, M NABIL, KC
MALLORY, JOHN A, SHAWNEE MISSION
MALONE, EUGENE M, HALSTEAD
MANAHAN, G EUGENE, LAWRENCE
MANI, M MANI, KC
MANLEY, JOSEPH W, SHAWNEE MISSION
*MANNING, ROBERT T, WICHITA
MANSFIELD, CARL M, KC
MANSOUR, BADIE S, WICHITA
MANTZ, FRANK A, SHAWNEE MISSION
MARCHBANKS, DONALD L, SALINA
MARR JR, JAMES C, COFFEYVILLE
MARSH, HENRY O, WICHITA
MARSHALL, B M, TOPEKA
MARSHALL, GEORGE O, COLBY
MARSHALL, GEORGE W, SALINA
MARSHALL, RONALD L, MANHATTAN
MARTIN, DANIEL C, MANHATTAN
MARTIN, EARL A, PARSONS
MARTIN, NORMAN L, KC
MARTIN, OLIVER L, SALINA
MARTIN, WILLIAM O, TOPEKA
MARTIN JR, ALBERT E, COFFEYVILLE
MARTIN JR, GLEN E, WICHITA
MARVIN, NORMAN G, KC
MARYMONT JR, JESSE H, WICHITA
MASER, GEORGE R, SHAWNEE MISSION
MASTERS, FRANCIS W, KC
MASTERSON, BYRON J, KC
MASTIO JR, GEORGE J, WICHITA
MATASSARIN, BENJAMIN M, WICHITA
MATASSARIN, FREDERICK W, WICHITA
MATHEWS, ROBERT MAJOR, SHAWNEE MISSION
MATHEWSON, HUGH S, KC
MATHIS, JERRY L, SALINA
MATTHEW, WILLIAM L, OLATHE
MATTICK, IRVIN H, HAYS
MATTIOLI, LEONE, KC
MAU, WALTER, TOPEKA
MAUCK, HAROLD C, STOCKTON
*MAWDSLEY, MICHAEL W, WICHITA
MAXFIELD, RUSSELL J, GARDEN CITY
MAXWELL, GORDON E, SALINA
MAXWELL, ROBERT A, SHAWNEE MISSION
MAY, KENNETH L, BONNER SPRINGS
MCALLASTER, WENDALE E, GREAT BEND
MCCANN, PATRICK E, FORT SCOTT
MCCANN, WILLIAM E, OLATHE
MCCARTER, DUANE K, TOPEKA
MCCARTHY, ROBERT P, KC
MCCAUGHEY, HUGH W, SHAWNEE MISSION
MCCLANAHAN, WARD A, WICHITA
MCCLELLAN, ERNEST L, WICHITA
MCCLELLAN, JOHN W, TOPEKA
MCCLINTOCK, EDWARD A, TOPEKA
MCCLURE, JAMES A, TOPEKA
MCCOLLUM, MARY B, LEAVENWORTH
MCCOLLUM, WILLIAM B, LEAVENWORTH
MCCOMAS JR, MARMADUKE D, CONCORDIA
MCCONNELL, ARCHIE B, BURLINGTON
MCCORMICK, EUGENE C, WELLINGTON
MCCOY, CHARLES P, WICHITA
MCCOY, CHARLES T, HUTCHINSON
MCCOY, RONALD, DODGE CITY
MCCRAE, SPENCER C, SALINA
MCCREIGHT, EUGENE J, LOGAN,NM
MCCUSKER, KEVIN T, KC
MCEACHEN, WILLIAM H, SHAWNEE MISSION
MCELHINNEY, CHARLES F, DODGE CITY
MCELROY, ROBERT T, TOPEKA
MCELROY, WILBUR J, ADDIS ABABA,ET
MCGOVERN, JAMES L, WELLINGTON
MCGUIRE, MICHAEL R, KC
MCGUIRE, WILLIAM F, WICHITA
MCGURK, THOMAS E, SHAWNEE MISSION
MCILVAIN, GUY B, CLAY CENTER
MCKEE, DICK B, PITTSBURG
MCKEE, LEO F, COTTONWOOD FALS
MCKEE, RICHARD S, LEAVENWORTH
MCKENNA, MICHAEL J, FORT SCOTT
MCKERRACHER, ROBERT O, DERBY
MCKIM, W LYNN, KINSLEY
MCKNIGHT, DAVID E, MANHATTAN
MCKNIGHT, ELLIS B, ALMA
MCLAIN, KENNETH, RANSOM
*MCMAHON, MERRI M, WICHITA
MCMULLEN, JOSEPH E, HUTCHINSON
MCNEIL, ELBERT D, MANHATTAN
MCVAY, R BRUCE, CLAY CENTER
MCWHERTER, LOTTIE B, SHAWNEE MISSION
MEBUST, WINSTON K, KC
MEDEARIS SR, DONALD N, KC
*MEADOWS, DONALD C, WICHITA
MEDINA, ANIBAL, HAYS
MEE, ADRIAN W, OLATHE
MEEK, GEORGE C, ARKANSAS CITY
MEEK JR, JOSEPH C, KC
MEEK, PALMER F, MANHATTAN
MEEKER II, BRUCE P, WICHITA
MEGIBOW, ALAN O, TOPEKA
MEHAFFY, ORVILLE A, BAXTER SPRINGS
*MEHRA, PROMILLA, WICHITA
MEIDEINGER, RICHARD, TOPEKA
MEIDINGER, RAY, HIAWATHA
MEKIS, JOHN, CHERRYVALE
*MELEAN, JAIME, WICHITA
MELENDRES, JUANITO M, SHAWNEE MISSION
MELIA JR, B JAMES, SHAWNEE MISSION
*MELVIN, DAVID B, WICHITA
MENAKER, JEROME S, WICHITA
MENDIONES, L MARLENE, WICHITA
MENDIONES, RUPERTO D, WICHITA
MENEHAN, H JAMES, WICHITA
MENENDEZ, JOSE O, SHAWNEE MISSION
MENEZ, CESAR V, SHAWNEE MISSION
MENKING, F W MANFRED, WICHITA
MENNINGER, KARL A, TOPEKA
MENNINGER, ROBERT G, TOPEKA
MENNINGER, ROY W, TOPEKA
MENNINGER, W WALTER, TOPEKA
*MERCADER, MARIO S, WICHITA
MERCER, JAMES B, SHAWNEE MISSION
MEREDITH, W TOM, WICHITA
MERKEL, EARL D, RUSSELL
MERRITT, JOE P, WICHITA
MERRITT, W HENRY, LEAVENWORTH
MERSHON, JAMES C, WICHITA
MESINA, ROLANDO R, KC
MEULBROEK, HARVEY J, WICHITA
MEYER, WARREN E, WICHITA
MEYERS, STEPHEN, GARDEN CITY
MICHELBACH, ALBERT P, WICHITA
MIGUELINO, OLIVER M, EMPORIA
MIH, ALEXANDER, CHANUTE
MILLER, ABRAHAM H, MANHATTAN
MILLER, CHARLES H, PARSONS
MILLER, CLYDE W, DOUGLAS
MILLER, DEAN M, PARSONS
MILLER, DON E, WICHITA
MILLER, EARL E, PITTSBURG
MILLER, ELDEN V, SALINA
MILLER, FRANKLIN R, WINFIELD
MILLER, HERBERT C, KC
*MILLER, LAWRENCE H, WICHITA
MILLER, MONTE B, RIALTO,CA
*MILLER, PHILIP A, WICHITA
MILLER, ROBERT E, GARDEN CITY
MILLIGAN, DONALD B, OLATHE
MILLS, CHARLES O, WICHITA

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MILLS JR, PHILIP E, TOPEKA
MIMOSO, JOSE J, DODGE CITY
MINER JR, PHILIP B, KC
MINNICK, CHARLES V, JUNCTION CITY
MIRZA, MEDO, WICHITA
MITCHELL, ALEX C, WICHITA
MITCHELL, JOHN C, SALINA
MITTLEMAN, FREDERICK S, TOPEKA
MITTS, ERNEST W, BONNER SPRINGS
MOODRELL, CAROL A, LAWRENCE
MODLIN, HERBERT C, TOPEKA
MOELLER, DONALD D, KC
MOFFAT, ROBERT E, KC
MOHLER, JACK M, ABILENE
MONTGOMERY, DAN, HALSTEAD
MONTGOMERY, THOMAS ALLEN, SABETHA
MONTGOMERY-SHRT, RUTH GERTRUDE, HALSTEAD
MOORE, CHESTER E, OAKLAND,AR
MOORE, DENNIS F, WICHITA
MOORE, HUGH C, TOPEKA
MOORE, JAMES E, CONCORDIA
MOORE, ROBERT, HOISINGTON
MOORE, ROBERT F, CANEY
MOORE, WAYNE V, KC
MOORHEAD JR, F ALLEN, NEODESHA
MORALES, AMALIA O, OSAWATOMIE
MORALES, OTTO E, OSAWATOMIE
MORAN, JEFFERY, TOPEKA
MORANTZ, ROBERT A, KC
MORCOS, ABDELMALAK E, SHAWNEE MISSION
MORFFI, RAUL R, KC
MORGAN, DAVID LLOYD II, OLATHE
MORGAN, DICK A, WICHITA
MORGAN, JAMES I, WICHITA
MORGAN, JOHN L, EMPORIA
MORGAN, NOVA L, HAYSVILLE
MORGAN III, LOUIS S, WICHITA
MORRIS, MERLE O, TOPEKA
MORRISON, IRA R, ATCHISON
MORRISON, RICHARD A, KC
MORRISON, RICHARD L, WICHITA
MORROW, THOMAS F, WICHITA
MORROW JR, J TARLTON, TOPEKA
MORTON, JOHN E, HALSTEAD
MORTON, ROSCOE F, ARKANSAS CITY
MOSELEY, JACK E, WICHITA
MOSER, ERNEST C, HOLTON
MOSER, M ROSS, HOLTON
MOSER, ROY H, HOLTON
MOSIER, STANLEY JAY, WICHITA
MOSIER, STEVEN J, MANHATTAN
*MOTEKALLEN, MOHAMMED, WICHITA
MOWERY, WILLIAM E, SALINA
MOWRY, GERALD L, MANHATTAN
MOY, JAMES T, WICHITA
MOYER, HERMAN J, DERBY
MOYER, JOHN O, TOPEKA
MUEHLBERGER, JAMES J, SHAWNEE MISSION
MUELLER, ARNOLD V, TOPEKA
MUELLER, J KENT, SHAWNEE MISSION
MUELLER, VERNETTE A, WICHITA
MULL, JOHN C, HUTCHINSON
MULLEN JR, CLIFFORD J, KC
MULLEN SR, CLIFFORD J, KC
*MULLINIX, JANICE M, WICHITA
MULLER, SAMUEL B, PITTSBURG
MURFITT, MALCOLM C, LINDSBORG
MURPHY, BARRY L, WICHITA
MURPHY, DUANE A, WICHITA
MURPHY, PAUL M, WICHITA
*MURRAY, KENT B, WICHITA
MYERS, JO ANN, TOPEKA
MYERS, ROBERT W, NEWTON
MYERS, W EUGENE, IOLA
MYERS JR, EARL B, INDEPENDENCE
MYRICK, MICKEY, HAYS

N

NABOURS, RICHARD O, TOPEKA
NACHTIGALL, ANDREW, NEWTON
NAIK, GOPAL V, PAOLA
NARCISO, VICENTE O, ABILENE
NASH, NEWMAN CURTIS, SCOTTSDALE,AZ
NASH, ROBERT A, SHAWNEE MISSION
NASON, HERBERT M, KC
NEEL, WILBUR B, HUTCHINSON
NEFF, JAMES R, KC
*NEGRON, ANGEL G, WICHITA
NEIBURGER, JAMES B, SHAWNEE MISSION
NEIGHBOR, ERNEST G, SHAWNEE MISSION
NEIGHBOR, ERNEST H, PITTSBURG
NEIGHBOR, GAYLORD P, KC
NEIGHBOR, RALPH M, EMPORIA
NEIL, ROY NEWTON, HAYS
NEIS, HARRY B, OSAGE BEACH,MO
NELSON, GERALD D, WICHITA
NELSON, JAMES N, TOPEKA
NELSON, PAUL L, CONCORDIA
NELSON, RICHARD O, LAWRENCE
NELSON, RUSSELL ALAN, WICHITA
NELSON, T EUGENE, FORT SCOTT
NELSON JR, GUST H, WICHITA
NEMMERS, DAVID J, JUNCTION CITY
NESMITH, LESLIE W, WICHITA
NEUENSCHWANDER, JOHN, HOXIE
NEUENSCHWANDER, JOHN RAND, HOXIE
NEUSCHAFER, DARREL R, HUTCHINSON
NEVINS, RICHARD L, LIBERAL
NEVITT, J RUSSELL, MORAN
NEWCOMB, WARD M, WICHITA
NEWCOMB, WARD M, HAYS
NEWKIRK, DARREL O, KC
NEWMAN, CLIFFORD B, PITTSBURG
NEWSOM, F CARTER, WICHITA
NEWTON, CHARLES R, PORTLAND,OR
NICE, G WILLIAM, TOPEKA
NICKELL, WENDELL K, SALINA
*NIEBLES, ANGEL, WELLINGTON
NIEDEREE, W CURTIS, GREAT BEND
NIEMAN, JOHN L, SHAWNEE MISSION
NIENSTEDT, JOHN F, SUN CITY,AZ
NIXON, RICHARD R, CONCORDIA
NIXON, WILLIAM A, WICHITA
NOHE, PHILIP C, KC
NORRIS, CHARLEY W, KC
*NORRIS, REBECCA M, WICHITA
NORRIS, ROBERT P, WICHITA
NORTH, DORIS G, WICHITA
NORTH, VICTOR, WICHITA
NORTON, ROBERT K, WICHITA
NOSTI, JUAN C, SHAWNEE MISSION
NOTHNAGEL, ARNOLD F, KC
NOVOTNY, PETER C, TOPEKA
*NOWLIN, NANCY S, WICHITA
NULL, WILLIAM G, SALINA
NUNEMAKER, MARION E, HUTCHINSON
NUNEZ, JULIAN, SHAWNEE MISSION
NYBERG, FREDRIK F, WICHITA
NYE, C ERIK, SHAWNEE MISSION
NYSTROM, CURTIS A, TOPEKA

O

O DONNELL, HAROLD F, SUN CITY,CA
O DONNELL JR, LEONARD A, WICHITA
O DONNELL SR, LEONARD A, WICHITA
O GRADY, JOSEPH A, HALSTEAD
OBANDO, GUILLERMO, SALINA
OBOURN, ROBERT L, TOPEKA
OBRYAN, JAMES J, SHAWNEE MISSION
OCHSNER, BRUCE B, WICHITA
OCONNELL, FRANK A, SHAWNEE MISSION
ODELL, MICHAEL L, SHAWNEE MISSION
ODENHEIMER, BURTRAM J, WICHITA
ODGERS, RODNEY K, PITTSBURG
ODONNELL, H ST CLAIR, ELLSWORTH
ODONNELL, HARRY E, JUNCTION CITY
OOONNELL, RICHARD H, CLAY CENTER
OELSCHLAGER, RONALD O, LAWRENCE
OH, SHI-HAN, WINFIELD
OHMAN, RICHARD J, DODGE CITY
OHMART, RICHARD V, OAKLEY
OKTAWIEC, DANUTA, SHAWNEE MISSION
OLD, JERRY L, ARKANSAS CITY
OLOFIELD, RAY W, KC
OLMO TERRON, NEFTALI, TOPEKA
OLNEY, ROBERT D, MANHATTAN
OLSEN, PHILLIP S, EL DORADO
OLSON, CARL E, LAWRENCE
OLSON, CLITUS W, GOODLAND
OLSON, ERWIN T, NEWTON
OLTMAN, THEODORE V, RILEY
ONEIL, ROBERT H, TOPEKA
OPENSHAW, CALVIN R, HUTCHINSON
ORCHARD, RICHARD A, LAWRENCE
OSBORNE, CONRAD C, WICHITA
OSGOOD, GEORGE M, SHAWNEE MISSION
OSHEA, JAMES G, JETMORE
OSIO, ANTONIO L, WICHITA
OSOBA, WILLIAM G, WICHITA
OTHMER, EKKEHARD, KC
OTOOLE, JAMES K, NEWTON
OVERHOLSER, NORMAN H, EL DORADO
OWEN, GARRY O, LAWRENCE
OWEN, LARUE W, WICHITA
OWEN, PERE A, WICHITA
OWENSBY, LINDELL C, CONCORDIA

P

PACE, JOHN O, PARSONS
PADGETT, WILLIAM L, WICHITA
PAGE, O VALE, PLAINVILLE
PAGE, RUTH G, WICHITA
PAINE, GEORGE E, HUTCHINSON
PALMER, DAVID L, WICHITA
PALMER, GERALD K, SALINA
PALMER, MARVIN M, LEAVENWORTH
PALMER JR, H C, LIBERAL
PANICHABHONGSE, SAMBHUNDH, RUSSELL
PARANJOTHI, SUBRAMONIAM P, PARSONS
PAROO, LILLIAN G, KC
PAROO, MANUEL P, KC
PAREKH, AJITKUMAR M, KANS CITY
PARK, CHAN H, KC
PARKER, HAROLD L, WICHITA
PARKER, ROBERT W, LEAVENWORTH
PARKER, WAYNE G R, EMPORIA
PARMAN, ROBERT D, TOPEKA
PARMLEY, CHARLES C, WICHITA
PARRA, MIGUEL O, KC
PARSI, MANUTCHEHR K, PITTSBURG
PASCHINO, ANTONIO R, KC
PASIMIO, ROGER S, COLUMBUS
PATEL, VINOD, TOPEKA
PATINO, EDGAR, TOPEKA
PATTERSON, ALICE M, LARNED
*PATTERSON, BRUCE W, WICHITA
PATTERSON, HAROLD L, LARNED
PATTERSON, JOHN R, SHAWNEE MISSION
PATTERSON, MICHAEL S, GARDEN CITY
PAULS, DANIEL N, PARSONS
* PAY, NORMAN T, WICHITA
PAYNE, ROBERT R, TOPEKA
PAZELL, JOHN A, KC
PEARCE, EUGENE W J, SHAWNEE MISSION
PEARCE, LUNETTA M, SHAWNEE MISSION
PECANA, MANUEL C, KC
PEDERSON, ARNOLD M, PLAINVILLE
PEORAZA, HERNANDO V, NEWTON
PEES, GERALD B, IOLA
PENCE, CHARLES O, WICHITA
PEFFLY, ELMER D, CHETOPA
PENN, GEORGE M, TOPEKA
PENNINGTON, KATHERINE, WICHITA
PENTECOST, RICHARD L, SHAWNEE MISSION
PEREIRA, WILLY G, ARKANSAS CITY
PERIDO, DOMINADOR T, ELKHART
PERKINS, JACK L, HUTCHINSON
PERRY JR, LAWRENCE L, KC
PETELIN, JOSEPH B, KC
*PETERIE, JERRY P, WICHITA
PETERS, DALE W, WICHITA
PETERS, GLENN R, KC
PETERSEN, A GENE, SHAWNEE MISSION
PETERSEN, GERALD O, SHAWNEE MISSION
PETERSON, DEAN L, TOPEKA
PETERSON, JACK T, MANHATTAN
PETERSON, ROBERT L, TOPEKA
PETERSON, VERNON J, TOPEKA
PETERSON JR, EVAN A, WATHENA
PETRIE, SAMUEL C, SHAWNEE MISSION
PETRIK, EDWIN L, TOPEKA
PETTEGREW, PAULINE K, SHAWNEE MISSION
PETTERSON, CECIL E, SYRACUSE
PETTERSON, O RUTH S, WICHITA
PETTIJOHN, WALTER J, RUSSELL
PFUETZE, BRUCE L, SHAWNEE MISSION
PFUETZE, KARL O, SHAWNEE MISSION
PFUETZE, ROBERT E, TOPEKA
PHILGREEN, DONALD E, OTTAWA
PHILLIPS, STEPHEN B, MANHATTAN
PHILLIPS, WARREN G, SHAWNEE MISSION
PHIPPS, JACK G, WICHITA
PIERCE, CHARLES F, TOPEKA
PIERCE, DONALD R, TOPEKA
PIERCE, GEORGE E, KC
PIERRON, GEORGE J, OLATHE
PIERSON, W WEIR, MCPHERSON
PILCHARD, WILLIAM A, KC
PINCOMB, ARTHUR L, OLATHE
PINSKER, JACOB A, WICHITA
PISCHKE, FRANK J, KC
PITMAN, WILL D, PRATT
PITTS, RONALD L, SHAWNEE MISSION
PLOWMAN, CARL W, JEWELL
PODREBARAC, LEONARD F, WICHITA
POGGI, RAYMOND G, TOPEKA
POGSON, GEORGE W, PITTSBURG
POKORNY, CHARLES, HALSTEAD
POL, P ALBERT, KC
POLING, TERRY L, WICHITA
POLLACK, SIMON, WICHITA
*POLLOCK, ANTHONY G A, WICHITA
POLLY, RICHARD E, TOPEKA
POLSON, ROBERT C, GREAT BEND
POOLE, BERNARD T, WICHITA
PORTER, DAVID M, KC
PORTER, GARRY L, WICHITA
PORTER, ROBERT O, TOPEKA
POTTER, ROBERT L, KC
POWELL, CAROL W, SHAWNEE MISSION
POWELL, KENNETH A, SHAWNEE MISSION
POWELL, WILLIAM R, TOPEKA
POWELL II, BENSON M, TOPEKA
POWERS, G ROBERT, KC
POWERS, HAROLD W, SUN CITY,AZ
POWERS, K DEAN, WICHITA
POWERS, WILLIAM F, SCOTTSDALE,AZ
*PRAEGER, MARK R, LAWRENCE
PRATT, WILLIAM L, LEAVENWORTH
PREMEIM, DELBERT V, NEWTON
PREMSINGH, NALINI G, KC

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PRENTISS, HAROLD, HALSTEAD
PRESTON, DAVID F, KC
PRESTON, RALPH R, TOPEKA
PRESTON, RICHARD, GREAT BEND
PRETZ, JAMES B, KC
PRICE, VAUGHAN C, MCPHERSON
PRICE JR, LAURANCE W, LAWRENCE
PRIETO, JORGE N, GARDNER
PRIETO, LUIS E, WICHITA
PROCHAZKA, OTTO F, LIBERAL
PROCTOR, ROBERT W, EL DORADO
PROKOP, BRADFORD S, TOPEKA
PRONKO, MICHAEL J, SHAWNEE MISSION
PROUD, G ONEIL, KC
PRUITT, JOHN C, WICHITA
PUGH, DAVID M, KC
PULIDO, QUINTON M, ELKHART
PULLMAN, NORMAN K, WICHITA
PURINTON, LEW W, WICHITA
PURVES, ROBERT K, WICHITA
PUTHENPURACKAL, GEORGE, WINFIELD
PUTNAM, LYLE B, WICHITA
PYLE, LUCIEN R, TOPEKA

Q

QAMAR, YUSUF, NEWTON
QUACKENBUSH, ROBERT P, ST JOHN
QUAISON, EMMELINE P, KC
QUENZER, RONALD W, PRATT
QUINN, CHARLES E, KC
QUINONES, ELADIO A, OSAWATOMIE

R

RABE, MELVIN A, LEAVENWORTH
RADOM, SANFORD B, SHAWNEE MISSION
RADOVANOV, RADMILA, NEWTON
*RAHBAR, AL A, WICHITA
RAHMAN, HAFIZ M A, ARCADIA,FL
RAINBOW, KATHRYN A, TOPEKA
RAMIREZ, AUGUSTO H, PITTSBURG
RAMSEY, BARTLETT W, TOPEKA
*RANDALL, GEORGE R, WICHITA
RANDLES, MICHAEL J, WICHITA
RANSDELL, EDGAR C, TOPEKA
RANSOM, JAMES H, TOPEKA
RASSA, REZA P, SALINA
RATE, ROBERT G, HALSTEAD
RATHBUN, EDWIN D, LIBERAL
RATNAM, DASARI V, OLATHE
RAUSA JR, FRANCISCO C, WICHITA
RAWCLIFFE JR, ROBERT A, WICHITA
*RAZEK, ZACK A, WICHITA
READ, WILLIAM T, COFFEYVILLE
REALS, WILLIAM J, WICHITA
REAZIN, WALTER L, WICHITA
RECKLING, FREDERICK W, KC
REDDY, B N, HILL CITY
REDDY, EASHWER K, KC
REDDY, NARAYAN C, SHAWNEE MISSION
REDDY, P JAGANNADHA, HILL CITY
REDDY, SATTI S, DODGE CITY
REDFORD, JOHN W B, KC
REECE, A THOMAS, GARDNER
REECE, ADELBERT S, GARDNER
REECE, CAROL A, GARDNER
REECE, RICHARD J, SALINA
REED, A J, WICHITA
REED, D CRAMER, WICHITA
REED, DAVID O, WICHITA
REED, JAMES S, WASHINGTON,DC
REED, RALPH R, LAWRENCE
REESE, JACK O, LIBERAL
REESE, JOHN L, LAWRENCE
REEVES, BRADFORD F, WICHITA
REEVES, CHARLES S, FORT SCOTT
REGIER, HENRY L, KC
REGIER, LADONNA M, COLBY
REIMER, DONALD R, TOPEKA
REINHARDT-WULF, TAISSIA L, GARDEN PLAIN
REINKING, VICTOR E, TOPEKA
REITZ, LELAND C, MANHATTAN
REITZ, ROGER P, MANHATTAN
REIVICH, RONALD S, SHAWNEE MISSION
RELIHAN, DONALD A, WICHITA
RELIHAN, FRANCIS H, SMITH CENTER
REMPEL, JOHN H, WICHITA
RENDON, HUMBERTO M, TOPEKA
*RENNEBOHM, ROBERT M, WICHITA
RENNER, MARION J, GODDARD
REPLOGLE, CHARLES B, GREAT BEND
RETTENMAIER, ALBERT J, KC
REUSCH, CLIFFORD S, TWIN FALLS,ID
REYES JR, FRANCISCO A, OTTAWA
REYMOND, RALPH O, TOPEKA
REYNOLDS, JEFFREY C, HAYS
REYNOLDS, LLOYD W, HAYS
RHOADS, JAMES P, WICHITA
RHODEN, CURTIS H, WICHITA
RHODES, IVAN E, WICHITA
RHODES, JAMES B, KC
RHODES, LOWELL M, WICHITA
RHODES, MARTIN L, KC
RICE JR, FREDERICK A, KC
RICH, ELDON S, NEWTON
RICHARDS, DENNIS D, CLAY CENTER
RICHARDS, JON F, PHILLIPSBURG
RICHARDSON, JAY L, ARKANSAS CITY
*RICHARDSON, J M, TOPEKA
RICHARDSON, STEWART F, WICHITA
RICK JR, GREGORY GP, KC
RIDER, JAMES W, ATCHISON
RIEDEL, ROBERT H, TOPEKA
RIEGER, ERNEST H, WICHITA
RIEKE, FRANK A, SHAWNEE MISSION
RIGLER, WILSON F, ARMA
RILEY, RAY B, KC
*RINDT, PHILLIP, WICHITA
RIORDAN, HUGH D, WICHITA
RISING, JESSE D, KC
RIZZA, ROBERT G, HALSTEAD
ROACH, NEIL E, HALSTEAD
ROBERTS, DANIEL K, WICHITA
ROBERTS, HOWARD E, TOPEKA
ROBERTS, LOUIS S, WICHITA
ROBERTS, RICHARD S, LAWRENCE
ROBERTS, WARREN E, TOPEKA
ROBERTSON, JOSEPH K, WICHITA
ROBINSON, DAVID B, TOPEKA
ROBINSON, DAVID W, KC
ROBINSON, EDGAR L, INDEPENDENCE
ROBINSON, G DONALD, WICHITA
ROBINSON, JOHN D, KC
ROBINSON, JOHN E, WICHITA
ROBINSON, RALPH G, KC
ROBINSON, ROBERT H, WICHITA
ROBISON, CORBIN E, LAWRENCE
ROBISON, JAMES T, KC
*ROBL, DAVID A, WICHITA
ROCERETO, PAUL V, TOPEKA
ROCHANAYON, PIRA, ELLIS
RODERICK, JAMES E, SALINA
RODRIGUEZ, RAUL G, KC
RODRIGUEZTOCKER, LILIA, WICHITA
ROEDER, ROBERT E, TOPEKA
ROMALIS, BRIAN E, WICHITA
ROMBOLD, CHARLES R, AUGUSTA
ROMEISER, REX S, SALINA
ROMONDO, STEVEN A, OLATHE
ROOK, LEE E, KC
RORABAUGH, DONALD C, ABILENE
ROSE, DONALD L, BELLA VISTA,AR
ROSE, GRAHAM C, MANHATTAN
ROSE, SHELBY D, WICHITA
ROSEN, DAVID, WICHITA
ROSENBERG, STANTON L, SHAWNEE MISSION
ROSENBERG, THOMAS F, WICHITA
ROSENTHAL, STANTON J, KC
ROSKOS, S RICHARD, TOPEKA
ROSS, DAVID K, ARKANSAS CITY
*ROSS, DENNIS LEE, WICHITA
ROSS, JACK L, TOPEKA
ROSSITTO, ANTHONY F, SAN FRANCISCO,CA
ROTH, ALAN E, KC
*ROTHSTEIN, TERRY B, PARSONS
*ROULEAU, DAVID N, NEWTON
ROVINSKI, HELEN T, TOPEKA
ROWLETT, JACK G, PAOLA
ROY, WILLIAM R, TOPEKA
RUBIN, HERBERT M, SHAWNEE MISSION
RUBIN JR, BEN, KC
RUBLE JR, JAMES L, OVERBROOK
RUCKER, CLEMENS, TOPEKA
RUEB, ANDREW E, SALINA
RUHLEN, JAMES L, OLATHE
RUIZ, CARLOS M, GREAT BEND
RUMOLD, MERVIN J, SHAWNEE MISSION
RUNNELS, JOHN B, TOPEKA
RUPP, RICHARD J, TOPEKA
RUSSELL, HOMER B, PEA RIDGE,AR
RUSSELL, PHILIP W, WICHITA
RUTH, WILLIAM E, KC
RUZICKA, LAWRENCE J, CONCORDIA
RYAN, EDWARD J, EMPORIA
RYAN, MICHAEL J, KC
*RYAN, THOMAS F, MANHATTAN
RYAN, W SCOTT, EMPORIA

S

SABIN JR, GEORGE M, WICHITA
SADIQ, SULEMAN, WICHITA
SAEED, MOHAMMAD A, WICHITA
SAFFO, KARL S, SHAWNEE MISSION
SALGADO, CARLOS R, WICHITA
SAMUEL, CHANOY C, WINFIELD
SANCHEZ, ROGELIO, TOPEKA
SANDBERG, CHRIS B, EL DORADO
SANDERS, J ALAN, LAWRENCE
SANDHU, PAUL S, COFFEYVILLE
SANTOSCOY, GILBERT S, WICHITA
SARGENT, JOSEPH D, TOPEKA
SATHYANARAYANA, SARASWATHI, SHAWNEE MISSION
SAUL, F WILLIAM, EMPORIA
SAVIN, MICHAEL A, KC
SAVIN, VIRGINIA J, KC
SAWKAR, LAXMIDAS A, SHAWNEE MISSION
SAYLER, JEROME, GREAT BEND
SAYLOR, EDWARD H, TOPEKA
SAYLOR, LESLIE L, TOPEKA
SAYLOR, MARK, TOPEKA
SAYLOR, STEPHEN, OTTAWA
SCALES, WILLIAM M, BLUE EYE,MO
SCAMMAN, W MIKE, TOPEKA
SCANLON JR, JAMES H, HADDAM,CT
*SCANLAN, TIMOTHY M, WICHITA
SCHAEFER, JOSEPH PETER, SHAWNEE MISSION
SCHAFFER, CLARENCE K, SANTA CRUZ,CA
SCHAUM, STEPHEN P, KC
SCHELLINGER, RICHARD P, EMPORIA
SCHERER, ALFRED L, ST JOHN
SCHIMKE, R NEIL, KC
SCHLACHTER, ERNEST R, WICHITA
SCHLEMMER, ROGER B, PITTSBURG
SCHLICHER, JOHN E, WICHITA
SCHILTZ, FRANCES, WICHITA
SCHLOERB, PAUL R, KC
SCHLOESSER, HARVEY L, TOPEKA
SCHLOTTERBACK, WILLIAM E, MANKATO
SCHLUETER, JOHN J, WICHITA
SCHMAUS, LYLE F, IOLA
SCHMIDT, HERBERT R, NEWTON
*SCHNELLE, JOACHIM, WICHITA
SCHNOEBELEN, RENE E, KINSLEY
SCHOPF, CLIFTON C, WICHITA
SCHOTLAND, EDWARD S, KC
SCHREPFER, ROSEMARY, KC
SCHROEDER, SYDNEY O, LAWRENCE
SCHROLL, JACK C, HUTCHINSON
SCHUETZ, PERRY N, GREAT BEND
SCHULTZ, JAMES E, COUNCIL GROVE
SCHULZ, MILLARD E, WICHITA
SCHWARTING, J STEVE, ABILENE
SCHWARTZ, EUGENE W, DODGE CITY
SCHWARTZ, V DEAN, WICHITA
SCHWEGLER, RAYMOND A, LAWRENCE
SCHWEGLER, RAYMOND A, SHAWNEE MISSION
SCHWORM, CURTIS P, KC
SCOTT, ALEX, JUNCTION CITY
SCOTT, CHESTER E, SALINA
SCOTT, DUANE L, BELLEVILLE
SCOTT, VINCENT L, WICHITA
SCOTT, WILLIAM H, WICHITA
SEAGO, CHARLOTTE L, LIBERAL
SEAMAN, LAUREN I, OLATHE
SEBREE, STEVEN G, SALINA
SEELEY, JAMES C, HOLTON
SEGERSON, JOHN A, TOPEKA
SEHOEV, JOAN, TOPEKA
SEITZ JR, JOSEPH E, ELLSWORTH
SEKAVEC, GORDON B, OAKLEY
SERERES, EDGAR P, KC
SERRANO, JOAQUIN, LA CROSSE
SETTLE JR, RUSSELL O, OLATHE
SETTLE SR, RUSSELL O, TOPEKA
SEVIER, SAMUEL M, TOPEKA
SHAAD, DOROTHY J, SHAWNEE MISSION
SHAFER, PRESTON J, WICHITA
*SHAH, MIAN, LARNED
*SHAH, MUKHTAR H, WICHITA
*SHAH, NASREEN, LARNED
SHAH, SHARFUDDIN, HALSTEAD
SHAW, JAMES W, WICHITA
SHAW, JOSEPH L, TOPEKA
SHAW, RICHARD C, WICHITA
SHAW JR, JAMES W, HUTCHINSON
SHEAFOR, DOUGLAS, TOPEKA
SHEARS, ROBERT N, HUTCHINSON
SHEFFER, KEITH O, OLATHE
SHEIKH, MASOOD A, INDEPENDENCE
SHELLITO, JOHN G, WICHITA
SHELTON, STEPHEN E, TOPEKA
SHEPARD, LEROY W, LARNED
SHEPHERD, GLEN R, CORONA DEL MAR,CA
SHEPPARD, ROBERT G, SMITH CENTER
SHERMAN, ROBERT P, KC
SHERWOOD JR, CLARENCE E, TOPEKA
SHIELDS JR, JAMES M, EL DORADO
SHIFLET, ALBERT W, WICHITA
SHIVEL, DAVID G, GREAT BEND
SHOFSTALL, WILLIAM H, SHAWNEE MISSION
SHRADER, DOYLE A, WICHITA
SIBALA, JUSTO L, KC
SIEGEL, ALBERT R, WICHITA
SIEMENS, RICHARD A, LYONS
SIFERS, EARL C, KC
SIFFORD, R LAWRENCE, WICHITA
SILER, EUGENE T, HAYS
SILLS, CHARLES T, NEWTON
SILVERGLAT, MICHAEL J, KC
SILVERS, ALVIN, KC
SIMMONS, WILLIAM C, WELLSVILLE
SIMPSON, J COLBERT, SALINA

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SIMPSON, TOM C, STERLING
SIMPSON, WILLIAM S, TOPEKA
SIMS JR, THOMAS J, SARASOTA,FL
SINCLAIR, ROBERT E, MANHATTAN
SINNING, GARY, HIAWATHA
SINGER, PHILIP A, KC,MO
SISK, PHILLIP B, TOPEKA
SKIBBA, RICHARD M, WICHITA
SLEEPER, CAROL A, WICHITA
SLEEPER, DONALD C, WICHITA
SLOO, MILO G, SALINA
SMILEY, WILLIAM A, JUNCTION CITY
SMITH, ALVIN L, WICHITA
SMITH, BRUCE G, ARKANSAS CITY
SMITH, DALE C, SHAWNEE MISSION
SMITH, DONALD J, SHAWNEE MISSION
SMITH, ERMA A, WICHITA
SMITH, HAROLD R, SALINA
SMITH, JOHN O, LARNED
SMITH, LEO A, TOPEKA
SMITH, NEWTON C, ARKANSAS CITY
SMITH, STEPHEN D, KC
SMITH, STEPHEN J, ARKANSAS CITY
SMITH JR, FLOYD L, COLBY
SMITH JR, WILLARD J, WICHITA
SNODELL, FIRMIN E, SHAWNEE MISSION
SNOOK, ROBERT RUFUS, MCLOUTH
SNOW, DONALD L, LANSING
SNOW JR, ARTHUR O, KC
SNOWBARGER, MARVIN D, EMPORIA
SNYDER, C JOHN, WINFIELD
SNYDER, GREGG M, WICHITA
SNYDER, HOWARD E, WINFIELD
SNYDER, THOMAS E, BILOXI,MS
SODOMSKY, MARTIN, TUCSON,AZ
SOELDNER, JAMES O, OLATHE
SOHLBERG JR, ROBERT, MCPHERSON
SOLOMON, HERMAN, WICHITA
SOLTZ, ROBERT A, WICHITA
SOMERS, MARVIN M, WICHITA
SONGER, HERBERT L, LINCOLN
SOUCEK, CHARLES O, KC
SPANN, RICHARD W, WICHITA
SPAULDING, JOHN S, KC
SPEARMAN, JESSE L, TOPEKA
SPEER, FREDERIC, SHAWNEE MISSION
SPEER, LELAND, KC
SPEER, LOUIS N, OTTAWA
SPENCER, MILLARD C, TOPEKA
SPENCER, WAYNE E, TOPEKA
SPIKES, MARION E, GARDEN CITY
STAFFORD, ROBERT W, HUTCHINSON
STANLEY, KENNETH E, WICHITA
STANLEY, REX C, PAOLA
STARK, JAMES R, WICHITA
STARKEY, JERALD L, RUSSELL
STECH, JOSEPH M, ANDALE
STECHSCHULTE, DANIEL J, KC
STEEGMANN, A THEODORE, INDIANAPOLIS,IN
STEELE, CLARENCE H, KC
STEICHEN, EDWARD F, LENDRA
STEIN, JOSEPH M, TOPEKA
STEIN, PAUL S, WICHITA
STEIN, ROBERT B, MANHATTAN
STEINER, ROBERT M, SHAWNEE MISSION
STEINKRUGER, VERLYN W, EMPORIA
STEINZEIG, ALFRED S, SHAWNEE MISSION
STEINZEIG, SHERMAN M, KC
STENSAAS, CARL D, HUTCHINSON
STEPHENS, CHARLES, MINNEOLA
STEPHENS, RONALD L, KC
STEPHENSON, LUCILLE C, ST FRANCIS
STEVENS, MILDRED J, GARNETT
STEVENS, PHILIP L, TONGANOXIE
STEVENS, ROBERT L, GARNETT
STEVENSON, CHARLES E, KC
STEVENSON, E KENT, SHAWNEE MISSION
STEWART, DAVID R, KC
STEWART, JACK T, WICHITA
STITT, RONALD W, KC
STOCK, KARL W, TOPEKA
STOCKWELL, MORGAN U, DODGE CITY
STOFER, BERT E, SUN CITY,AZ
STOFFER, ROBERT P, HALSTEAD
STOKES, ROBERT LEE, KANSAS CITY,MO
STOLZ, ELMER G, WICHITA
STONE, G REX, MANHATTAN
STONE, GRANT C, ATTICA
STOPPLE, JOHN A, OLATHE
STOSKOPF, LAWRENCE E, WICHITA
STOUT, JAMES M, HUTCHINSON
STOUT, NILES M, LYNDON
STRAHM, WAYMER J, INDEPENDENCE
STRANGE, MICHAEL, WICHITA
STREET, DAVID E, WICHITA
STREHLOW, CHESTER H, OTTAWA
STRICK, FRANK J, KC
STRUTZ, WILLIAM C, LEAVENWORTH
STRYKER JR, HENRY B, CONCORDIA
STRYKER, TERRY M, WICHITA

STUBBLEFIELD, CHARLES T, KC
STUCKY, DEAN E, MEDICINE LODGE
STUMP, HARL G, HAYS
*SUERO, JESUS T, WICHITA
SUGAR, ROBERT L, SHAWNEE MISSION
SULLIVAN, CORNELIUS J P, WICHITA
SULLIVAN, LEONARD L, WICHITA
SULLIVAN JR, HENRY B, SHAWNEE MISSION
SUMMERVILLE, WARD W, SHAWNEE MISSION
SUMNER, JOYCE R, HUTCHINSON
SUMNER, MARION M, HUTCHINSON
SUMNER, RALPH N, FREDONIA
SURFACE, GARDNER A, ELLIS
SUTTON, RICHARD D, TOPEKA
SUTTON, ROBERT E, OTTAWA
SUTTON JR, RICHARD L, SHAWNEE MISSION
SVOBODA, CHARLES R, CHAPMAN
SWAN, MAJOR MARTIN, GREAT BEND
SWANN, CLAIR L, RUSSELL
SWARTZ, WARREN E, PARSONS
SWEENEY, JAMES G, WICHITA
SWISHER, WILLIAM C, WICHITA
SWOGGER JR, GLENN, TOPEKA

T

TAKAHASHI, TETSURO, TOPEKA
TAN, POLY, HALSTEAD
TANODE JR, VALENTIN T, NEWTON
TANGCHUPONG, CHANTRASIRI, PARSONS
TANGCHUPONG, SAROHO, PARSONS
TAPPEN, DANIEL L, TOPEKA
TARGOWNIK, KARL K, TOPEKA
TARNOWER, WILLIAM, TOPEKA
*TATAATI, DANIEL A, WICHITA
TAYIEM, ABDEL K, ATCHISON
TAYLOR, ELMER W, SEDAN
TAYLOR, ELWYN J, HUTCHINSON
TAYLOR, RICHARD J, WICHITA
TAYLOR, THOMAS F, SALINA
TAYLOR, THOMAS L, KC
TEARE, MAX E, GARDEN CITY
TEJANO, NEDNILO A, HALSTEAD
TEMPERO, STEPHEN J, TOPEKA
TEMPLETON, ARCH W, KC
*TERRY JR, DANIEL W, WICHITA
TETZLAFF, ARCHIBALD D A, SHAWNEE MISSION
TEUSINK, J PAUL, TOPEKA
THAOA, NARONGSAK K, HAYS
THELEN, J CHRISTINE, WICHITA
THERDU, LEONA F, KC
THIEMANN, A H, D.O., SUBLETTE
THOMAS, JAMES H, KC
THOMAS, R CULLEN, PRATT
THOMAS, THOMAS V, KC
THOMPSON, DANIEL M, WICHITA
THOMPSON, DANNIE M, KC
THOMS, NORMAN W, TOPEKA
THORNTON JR, FOXHALL P, CONCORDIA
THORPE, FRANCIS A, PRATT
*THOMSEN, STEVEN T, LAWRENCE
THURSTON, DAVID E, TOPEKA
TICE, GALEN M, SHAWNEE MISSION
TIEMANN, WILLIAM H, MANHATTAN
TIHEN, EDWARD N, WICHITA
TIHEN, HENRY N, WICHITA
TILLER, GEORGE R, WICHITA
TILLOTSON, DON R, ULYSSES
TILTON, FRANK M, WICHITA
TINKER, ROBERT C, WICHITA
TINTEROW, MAURICE M, WICHITA
TIPPIN JR, ERNEST E, WICHITA
TIVORSAK, ARKOM, ATCHISON
TOALSON, WILLIAM B, KC
TOCKER, ALFRED M, WICHITA
TOMPKINS, CARL D, NEWTON
TONN, GERHART R, WICHITA
TOTTEN, FREDERICK E, LAWRENCE
TOVAR, MANUEL H, HAYS
TOZER, RICHARD C, TOPEKA
TRACY, TERRY A, WICHITA
TRAVIS, JOHN W, TOPEKA
TREES, CLYDE B, TOPEKA
TREES, DONALD P, WICHITA
TREGER, NEWMAN V, TOPEKA
TREKELL, WILLIAM V, DODGE CITY
TRETBAR, HARVEY A, WICHITA
TRETBAR, LAWRENCE L, SHAWNEE MISSION
*TREWEEKE, MICHAEL W, WICHITA
TRIMBLE SR, DAVID P, EMPORIA
TRIMBLE, DAVID P JR, OLATHE
TROPP, ARNOLD L, SHAWNEE MISSION
TROTTER, CORKY R G, MINNEOLA
TRUEWORTHY, ROBERT C, KC
TRUITT, WILLIAM ROY, TOPEKA
TUCKER, SHERIDAN G, KC
TUONG, TRAN M, AUGUSTA
TURNER, JOHN W, GARDEN CITY
TUTERA, GINO, SHAWNEE MISSION
TWEET, FREDERICK A, PITTSBURG
TWEITO, DAVID H, HUTCHINSON
TYLER, WILLIAM F, COFFEYVILLE

U

UBELAKER, ERNEST J, SOUTH HAVEN
UHLIG, PAUL J, WICHITA
UHR, NATHANIEL, TOPEKA
UNDERWOOD, CHARLES C, EMPORIA
UNREIN, ROBERT J, GREAT BEND
UY, WILSON D, COFFEYVILLE

V

VAKAS, JOHN L, COFFEYVILLE
VALK, WILLIAM L, KC
VAN DER VLIET, STEPHEN K, WICHITA
VAN THULLENAR, PHILIP A, KC
VANDE GARDE, LARRY D, TOPEKA
VANDER VELDE, STANLEY L, EMPORIA
VANNAMAN, DONALD D, SHAWNEE MISSION
VARGHESE, GEORGE, KANSAS CITY
VATS, TRIBHAWAN S, KC
VAUGHAN, DONNA A, NEWTON
VILLANUEVA, CESAR L, SHAWNEE MISSION
VIN ZANT, LARRY E, WICHITA
*VINZANT, WHITNEY L, WICHITA
VISWANATHAN, BYRAVAN, TOPEKA
VOGT, VERNON W, NEWTON
VOLKMANN II, HARLEY W, MANHATTAN
VONGKASEMSIRI, SUNAN, NEWTON
VON LEONROD JR, GEORGE, OIGHTON
VON RUDEN, WILLIAM J, HUTCHINSON
VOORHEES, CARROLL D, LEAVENWORTH
VOORHEES, GORDON S, LEAVENWORTH
VOPAT, RICHARD L, KC
VORHEES, VICTOR J, WICHITA
VOTAPKA, WILLIAM L, STOCKTON
VOTH, DOUGLAS W, WICHITA
VOTH, HAROLD M, TOPEKA
VRANEY, GEORGE, HALSTEAD

W

WADDELL, BILL D, KC
*WADUD, ABDUL, WICHITA
WADE, THEODORE E, LIBERAL
WAGENBLAST, HOWARD R, SALINA
*WAKIM, PAUL E, WICHITA
WAGGONER, FRANKLIN E, BONNER SPRINGS
WALDORF JR, MELVIN H, GREENSBURG
WALKER, JACK D, KC
WALKER, MAURICE A, KC
WALKER, NELLIE G, LEES SUMMIT,MO
WALKER, WILLIAM H, ESKRIDGE
WALKER, WILLIAM K, SEDAN
*WALKER, WILLIAM L, WICHITA
WALLACE, LEO F, TOPEKA
WALLACE, ROBERT W, WICHITA
WALLACE JR, WAYNE O, ATCHISON
WALLACE SR, WAYNE D, ATCHISON
WALLS, WILLIAM J, TOPEKA
WALSH, EUGENE A, ONAGA
WALSH, RICHARD A, WICHITA
WALSH, THOMAS E, ONAGA
WALTERS, BYRON W, LAWRENCE
WALTON, PHILIP O, HIAWATHA
WALZ, ROYCE C, TOPEKA
WALZ, THOMAS J, ST FRANCIS
WANG, SIDNEY W, SHAWNEE MISSION
WARD, HOWARD N, TOPEKA
WARD, JAMES A, BELLEVILLE
WARD, ROBERT L, STAFFORD
WARE, LUCILE M, TOPEKA
WARREN, LINDA O, WASHINGTON
WARREN, LLOYD P, WICHITA
WARREN, ROGER O, HANOVER
WARREN, WIRT A, WICHITA
WARREN JR, JOHN W, WICHITA
WATERS, CLARENCE N, SALINA
WATERS III, CHESTER H, KC
WATTS, HARRY E, HAYS
WAXMAN, DAVID, KC
WAYLAND, THORNTON L, NASHVILLE
WEARE, MARY E, SHAWNEE MISSION
WEAVER, J ROBERT, WICHITA
WEAVER, JACK D, WICHITA
WEAVER, JEROME A, SHAWNEE MISSION
WEAVER, WALTER O, TOPEKA
WEBB, JAMES R, SHAWNEE MISSION
WEBER, CLARENCE J, SALINA
WEBER, DARRELL J, TOPEKA
WEBER, ROBERT W, SALINA
WEBER, ROY R, NEWTON
WEBER, WALLACE N, HAYS
*WEBSTER, BOBBY W, WICHITA
WEBER JR, HUGO P, WICHITA
WEDDLE, DOUGLAS P, FORT SCOTT
WEDEL, KENNETH D, MINNEAPOLIS
WEDEL, KERMIT G, MINNEAPOLIS
WEDIN, PAUL H, WICHITA
WEIGAND, JOEL T, WELLINGTON
WEIGEL, JOHN W, KC
WEIPPERT, EDWARD J, WICHITA
WEIR, WILLIAM C, ERIE

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WELCH, JACK W, HALSTEAD
WELCH, LAUREN K, WICHITA
WELCH, MARTIN H, WICHITA
WELL, MICHAEL A, LAWRENCE
WELLS, ALVIN Y, WINFIELD
WELLS, BRUCE W, WINFIELD
WELLS, MAX W, WINFIELD
WELLSHEAR, CHARLES C, WICHITA
WELTMER, ROGER P, BELOIT
WENINGER, JOHN H, WICHITA
WERNER, WILLARD F, TRIBUNE
WERTH, CLAUDE J, KC
WERTZBERGER, JOHN, LAWRENCE
WESBROOK, CLYDE W, TRAVIS AFB,CA
WESCOE, W CLARKE, NEW YORK,NY
WEST, C OMER, KC
WEST, RAY A, WICHITA
WEST, WILLIAM T, WICHITA
WHALLON, JACOB T, WICHITA
WHEELER, PINCKNEY R, WICHITA
*WHITAKER, JAMES A, WICHITA
WHITAKER, REN R, OBERLIN
WHITE, CHARLES L, GREAT BEND
WHITE, CHARLES M, WICHITA
WHITE, DONALD C, COFFEYVILLE
WHITE, FAGAN N, RUSSELL
WHITE, HARRY H, TOPEKA
WHITE, JOHN P, PARSONS
WHITE, R BURNLEY, WINFIELD
WHITE, THADDEUS H, MANHATTAN
WHITE II, BENJAMIN E, EL DORADO
WHITEHEAD, RICHARD E, SHAWNEE MISSION
WHITLEY, DOUGLAS M, SHAWNEE MISSION
WIEGMAN, HUGH A, HAYS
WIENS, J WENDELL, NEWTON
WIENS, PETER K, NESS CITY
WIGGINTON, GERALD O, O.D., SHAWNEE MISSION
WIGGS, JAMES W, GREAT BEND
WIKSTEN, VERNON C, TOPEKA
WILCHINS, LAWRENCE J, WICHITA
WILCOX, DONALD E, TOPEKA
WILCOX JR, HOWARD L, HAYS
WILCOX SR, HOWARD L, LAWRENCE
WILDER, LOWELL W, WICHITA
WILEY, HORACE M, GARDEN CITY
WILEY, JOHN H, SHAWNEE MISSION
WILLIAMS, CHARLES L, WICHITA
WILLIAMS, EVAN ROBERT, DODGE CITY
WILLIAMS, GENE V, EL DORADO
WILLIAMS, HOMER J, OSAGE CITY
WILLIAMS, HOWARD V, TOPEKA
WILLIAMS JR, STERLING B, KC
WILLIAMSON, JOHN A, KC
WILSON, JAMES W, TOPEKA
WILSON, L BARRICK, SHAWNEE MISSION
WILSON, MARVIN H, TOPEKA
WILSON, ROBERT B, SHAWNEE MISSION
WILSON, ROBERT L, VALLEY CENTER
WILSON, SLOAN J, KC
WINBLAD, JAMES N, WINFIELD
WINCHELL, H H FORSYTH, WICHITA
WINCHESTER, EUGENE B, WICHITA
WING, NANCY J, TOPEKA
WINTER JR, CALVERT J, KC
WISDOM, JAY K, WICHITA
WISNER JR, HARRY J, WICHITA
WITTMANN, ALBERT F, WICHITA
WOLF, CURTIS V, LYONS
WOLF, KARL T, KC
WOLFE, FREDERICK, WICHITA
WOLFE, JOHN E, HUTCHINSON
WOLFENBARGER, KEITH A, HOISINGTON
WOLFF, FREDERICK P, PRATT
WOLKOFF, A STARK, KC
WOLLMANN, MARTIN, LAWRENCE
WONG, BERT Y, KC
WONG, NORMUND, TOPEKA
WOOD, DOUGLAS H, PITTSBURG
WOOD, GARY B, WICHITA
WOOD, ROBERT D, HORTON
WOODARD, LAWRENCE E, WICHITA
WOODHOUSE, CHARLES L, WICHITA
WOODS, HUGH J, SMITH CENTER
WOODS, ROBERT P, TOPEKA
WOOLLCOTT JR, PHILIP, TOPEKA
WORTHINGTON, J MACK, WICHITA
WORTMAN, JACK A, HUTCHINSON
WRAY, ALEXANDER J, WICHITA
WRIGHT, KENDALL M, EMPORIA
WRIGHT, M ERIK, LAWRENCE
WRIGHT, STANLEY E, JUNCTION CITY
WRIGHT JR, ROBERT W, KC
WULFF, EDWIN T, ATCHISON
WURSTER, GEORGE R, SHAWNEE MISSION

Y

YAGHMOUR, TALAAT E, PITTSBURG
YE, RICHARD C, SHAWNEE MISSION
YEOMANS, RONALD N, OLATHE
YODER, EMERSON O, DENTON
YODER, VERNON E, HESSTON
YOHE, RUTH M, SHAWNEE MISSION
YOUMANS, ROGER L, AFRICA,
YOUNG, CHARLES H, ATCHISON
YOUNG, JOHN W, SHAWNEE MISSION
YOUNG, THEODORE E, TOPEKA
*YOUNGBERG, DEAN I, WICHITA
YOUNGSTROM, KARL A, SHAWNEE MISSION
YULICH, JOHN O, KC

Z

ZABEL, KENNETH P, PITTSBURG
ZACHARIAS, CARL KURT, DODGE CITY
ZACK, ASHLEY S, SHAWNEE MISSION
ZAREMSKI, SHERMAN C, SHAWNEE MISSION
ZARNOW, HILARY, WICHITA
ZEE, HUGO J, COLUMBUS,GA
ZELLER, MYRON J, GARDEN CITY
ZIEGLER, DEWEY K, KC
ZIMMERMAN, DANIEL D, SHAWNEE MISSION
ZIMMERMAN, KENNETH D, WICHITA
ZIMMERMAN, WILLIAM H, TOPEKA
ZINN, THOMAS W, KC
ZONGKER, PHILIP E, WICHITA
ZORNOZA, JESUS, KC

* Probationary members.

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Medical Specialty Codes as Recognized by the AMA and Used in This Directory

ADM	Administrative Medicine
A	Allergy (sub-specialty of Internal Medicine)
ANES	Anesthesiology
AM	Aviation Medicine (special field of Preventive Medicine)
CD	Cardiovascular Disease (sub-specialty of Internal Medicine)
CHP	Child Psychiatry (sub-specialty of Psychiatry)
CLG	Clinical Geronotology
CP	Clinical Pharmacology
CRS	Colon and Rectal Surgery
DR	Diagnostic Roentgenology (special field of Radiology)
D	Dermatology
EM	Emergency Medicine
END	Endocrinology
ENT	Ear, Nose and Throat
FOP	Forensic Pathology (special field of Pathology)
FP	Family Practice
GE	Gastroenterology (sub-specialty of Internal Medicine)
GPM	General Preventive Medicine (special field of Preventive Medicine)
GS	General Surgery
GEN	Genetics
GER	Geriatrics
GYN	Gynecology
HEM	Hematology
HM	History of Medicine
ID	Infectious Diseases
IM	Internal Medicine
IE	Insurance Examination
ND	Neoplastic Diseases
NEP	Nephrology
NM	Nuclear Medicine
NS	Neurological Surgery
N	Neurology
NP	Neuropsychiatry
OBG	Obstetrics and Gynecology
OM	Occupational Medicine (special field of Preventive Medicine)
OPH	Ophthalmology
ORS	Orthopedic Surgery
OST	Osteopathy
OTO	Otolaryngology
PATH	Pathology
PD	Pediatrics
PDA	Pediatric Allergy (sub-specialty of Pediatrics)
PDC	Pediatric Cardiology (sub-specialty of Pediatrics)
PDH	Pediatric Hematology (sub-specialty of Pediatrics)
PDN	Pediatric Neurology (sub-specialty of Pediatrics)
PM	Physical Medicine and Rehabilitation
PS	Plastic Surgery
P	Psychiatry
PYM	Psychosomatic Medicine
PH	Public Health (special field of Preventive Medicine)
PUD	Pulmonary Diseases (sub-specialty of Internal Medicine)
R	Radiology
RES	Resident
RHU	Rheumatology
SH	School Health
TR	Therapeutic Radiology (special field of Radiology)
TS	Thoracic Surgery
U	Urology
OO	Retired
*	Probationary Member

Physician Distribution by Cities

EXPLANATION OF CODES USED IN THIS SECTION

Line 1: Doe, John R., 1234 Oak St., 67052
(Name) (Street Address) (Zip Code)

Line 2: (654-2222)
(Telephone Number)

Line 3: 33 M 1902 58 FP
(Year of Birth) (Sex) (Medical School) (Year of Graduation) (Specialty)

Telephone area code follows city name. * Probationary Members

ABILENE—913
(Dickinson County Society)

BERKLEY, DON H, 1200 NORTH BRADY,67410
263-4131
35 M 1902 61 FP
BIGGS, DENNIS, 515 NE 10TH,67410
263-7190
48 M 1902 74 FP
BUTCHER, ARCHIE W, 303 HILLTOP DR,67410
-
01 M 1902 35 OO
CHAFFEE DEAN C, 1200 N BRADY, 67410
263-4131
11 M 1902 44 FP
COLEMAN, GARY W, 515 NE 10TH,67410
263-7190
46 M 1902 72 FP
HEINS, LAWRENCE G, RR #5,67410
-
97 M 4102 21 OO
MOHLER, JACK M, 1200 NORTH BRADY,67410
263-4131
32 M 1902 61 FP
NARCISO, VICENTE O, 515 NE 10TH ST,67410
263-2253
45 M 74810 68 GS
RORABAUGH, DONALD C, PROFESSIONAL BLDG,67410
263-4131
33 M 1902 58 FP
SCHWARTING, J STEVE, 515 NE 10TH,67410
263-7190
46 M 3401 72 FP

ALMA—913
(Pottawatomie County Society)

MCKNIGHT, ELLIS B, ,66401
765-3317
03 M 1902 33 OD

ALTAMONT—316
(Labette County Society)

JACKSON, VICTOR L, BOX 467,67330
784-5318
20 M 2105 50 FP

ANDALE—316
(Sedgwick County Society)

STECH, JOSEPH M, ANDALE CLINIC,67001
445-2152
27 M 3006 56 FP

ANTHONY—316
(Tri-County Society)

ANTRIM, PHILIP J, 1101 E SPRING, 67003
842-5144
15 M 1902 42 FP
HALL, JERRY O, 1101 E SPRING,67003
842-5144
31 M 3901 56 FP

ARKANSAS CITY—316
(Cowley County Society)

ALVAREZ, NORBERTO, 112 E CENTRAL,67005
442-4850
27 M 27501 53 FP
AUCAR, ALFREDO, BOX 1105,67005
442-1710
23 M 27501 53 OTO
BRALEY, JACK A, O,O,, 1501 N 7TH,67005
442-6660
29 M 1875 68 IM
CAMPBELL, GARLAND L, 201 A C OFFICE BUILDING,67005
442-1350
13 M 1902 40 U
FARRAR JR, WILLIAM H, 2508 EDGEMONT,67005
442-8200
44 M 1902 70 IM
GREEN, LAWRENCE C, 112 EAST CENTRAL AV,67005
442-4850
43 M 3901 69 FP
HILL, JAMES E, 2508 EDGEMONT DR,67005
442-4300
09 M 1902 34 OPH
HINSHAW, EDGAR O, RT #3,67005
442-1273
15 M 1902 51 R
HOWARD II, W ROBERT, 2508 EDGEMONT,67005
442-1710
45 M 1902 71 ENT
MEEK, GEORGE C, 323 NORTH SUMMIT,67005
442-2100
05 M 1902 32 FP
MORTON, ROSCOE F, AC MEDICAL & SURG CLINIC,67005
442-2450
13 M 401 42 IM
OLD, JERRY L, 323 NORTH SUMMIT,67005
-
49 M 1902 74 FP
PEREIRA, WILLY G, 2508 EDGEMONT DR,67005
442-8540
39 M 73701 67 IM
RICHARDSON, JAY L, 2508 EDGEMONT DRIVE,67005
442-4150
38 M 1902 65 GS
ROSS, DAVID K, 232 NORTH SUMMIT,67005
-
48 M 1902 75 FP
SMITH, BRUCE G, 115 E RADIO LANE,67005
442-5600
20 M 1902 44 IM
SMITH, NEWTON C, MEEK STENSAAS CLINIC,67005
442-2100
21 M 3901 45 FP
SMITH, STEPHEN J, 115 HILLSIDE,67005
442-5600
45 M 1902 71 FP

ARMA—316
(Crawford County Society)

RIGLER, WILSON F, 511 WASHINGTON,66712
347-8619
42 M 1803 69 FP

ASHLAND—316
(Iroquois County Society)

LEE, JOSEPH R, ,67831
635-2724
25 M 24351 50 FP

ATCHISON—913
(Atchison County Society)

BOSSE, FRANK K, 1301 RIVERVIEW DRIVE,66002
-
09 M 2802 33 OO

BRADY, CHARLES S. 111 N 5TH ST.66002
367-1232
11 M 3006 38 GS
BRIBACH, EUGENE J. 125 1/2 N 5TH.66002
367-0225
83 M 2802 05 00
BROWN, ROBERT O. 1400 NORTH 2ND.66002
367-1922
14 M 1902 44 FP
BURKE, JOSEPH V. 1301 N 3RD.66002
367-5496
35 M 3006 66 GS
FAST, ROBERT E. 1225 N 2ND.66002
367-0362
48 M 1902 74 OBG
FAST, W SPENCER, RAMSAY MED CLINIC.66002
367-0362
11 M 3006 39 FP
GROWNEY, JOHN T. 801 ATCHISON.66002
367-5020
37 M 3006 63 FP
HART, LAWRENCE E. 1412 N 2ND.66002
367-5054
32 M 1902 64 FP
MORRISON, IRA R. 825 N 10TH.66002
367-4396
07 M 1611 36 IM
RIDER, JAMES W. 1225 N 2ND ST.66002
367-0362
47 M 2803 73 FP
TAYIEM, ABDEL K. 1225 N 2ND.66002
367-0362
43 M 33002 68 GS
TIVORSAK, ARKOM. PO BOX 328.66002
367-2131
40 M 89101 68 R
WALLACE JR, WAYNE O. 1301 N THIRD.66002
367-7300
36 M 2803 65 FP
WALLACE SR, WAYNE O. 1301 N THIRD.66002
367-5434
12 M 1902 37 FP
WULFF, EDWIN T. 111 N 5TH.66002
367-5033
07 M 2834 36 FP
YOUNG, CHARLES H. 1301 N 3RD.66002
367-4053
23 M 1902 53 FP

ATTICA—316
(Tri-County Society)

STONE, GRANT C. 215 W AVE D.67009
254-7219
08 M 5605 35 FP

ATWOOD—913
(Northwest Kansas Society)

HENNEBERGER, CHARLES E. 234 NORTH 7TH.67730
–
86 M 4101 10 00

AUGUSTA—316
(Butler-Greenwood Society)

ANDERSON, DALE W. 209 D WEST 7TH STREET.67010
775-5432
30 M 1902 55 FP
BARBER, JAMES L. AUGUSTA PLAZA.67010
775-5432
31 M 1902 57 FP
ROMBOLO, CHARLES R. RT 1.67010
–
99 M 1606 24 00
TUONG, TRAN M, AUGUSTA PLAZA, 67010
775-5432
39 M 65 GP

BAXTER SPRINGS—316
(Cherokee County Society)

ALQUIST, VERYL D. 21ST & FAIRVIEW.66713
623-4942
17 M 1902 42 GS
CHUBB, RICHARD M. 445 EAST 10TH.66713
856-2444
29 M 1606 54 FP
CLAIBORNE, RICHARD H. 2201 MILITARY AVE.66713
856-3535
27 M 1902 53 FP
MEHAFFY, ORVILLE A. 411 E 11TH.66713
856-2383
42 M 1902 69 FP

BELLEVILLE—913
(Republic County Society)

BEIDERWELL, PAUL L. 2703 M ST.66935
–
08 M 3901 38 FP
CHANEY, ERNIE J. PO BOX 250.66935
527-2237
27 M 1902 56 FP
DOUBEK, HERBERT O. 2316 G ST.66935
527-2237
28 M 1902 56 FP
SCOTT, DUANE L. BELLEVILLE CLINIC.66935
527-2217
34 M 1902 60 FP
WARD, JAMES A. 1206 18TH ST.66935
527-2217
34 M 1902 58 FP

BELOIT—913
(Mitchell County Society)

DOBRATZ, ROBERT A. 310 W 8TH.67420
738-2246
24 M 1902 52 FP
DRAKE, DOUGLAS J. MEDICAL CENTER.67420
738-2246
43 M 1902 71 FP
KLENDA JR, MARTIN B. BELOIT MED CENTER.67420
738-2246
38 M 1643 63 GS
LONEY, JOHN M. 310 WEST 8TH.67420
738-2246
50 M 1902 74 IM
WELTMER, ROGER P. 112 W MAIN.67420
738-2574
18 M 1902 44 U

BLUE RAPIDS—913
(Northeast Kansas Society)

LAWLESS, HAROLD L. 607 LINCOLN.66411
226-7202
29 M 702 54 FP

BONNER SPRINGS—913
(Wyandotte County Society)

MAY, KENNETH L. 122 N NETTLETON.66012
422-2020
20 M 1902 51 FP
MITTS, ERNEST W. 122 N NETTLETON.66012
422-2020
22 M 1902 51 FP
WAGGONER, FRANKLIN E. 122 NORTH NETTLETON.66012
422-2020
26 M 1902 61 FP

BUHLER—316
(Reno County Society)

FRIESEN, ORLANDO J. 107 W 2ND.67522
543-2330
27 M 1902 56 FP

BURLINGTON—316
(Flint Hills Society)

MCCONNELL, ARCHIE B. .66839
90 M 3901 15 00

CALDWELL—316
(Tri-County Society)

KINNAN, L F, 523 S MARKET, 67022
845-6422
18 M 3901 42 FP

CANEY—316
(Southeast Kansas Society)

MOORE, ROBERT F, 4TH & MCGEE,67333
879-2135
28 M 1902 56 FP

CEDAR VALE—316
(Southeast Kansas Society)

COHNBERG, ROSELLEN E, PO BOX 398,67024
758-2266
22 F 2802 47 FP

CHANUTE—316
(Southeast Kansas Society)

ABBUEHL, OON R, 505 SOUTH PLUMMER,66720
431-2500
18 M 1902 44 S
ASHLEY, SAMUEL G, 505 SOUTH PLUMMER,66720
431-2500
16 M 1902 43 FP
BAKER, HENRY K, 1501 WEST 7TH,66720
431-1600
08 M 1606 35 S
BURKMAN, REUBEN J, 1501 W 7TH,66720
431-9310
28 M 1902 54 FP
OICK JR, HENRY J, 1501 W 7TH,66720
431-9310
27 M 1902 58 FP
GEHRT, EARL B, 1101 S LARSON,66720
431-2500
32 M 1902 62 FP
HASKINS, ROBERT J, 505 S PLUMMER,66720
431-2500
46 M 1902 74 FP
HUDSON, JAMES R, 616 HILLSIDE,66720
431-4000
37 M 1902 63 R
KIHM, ALBERT A, 505 S PLUMMER,66720
431-2500
27 M 1902 55 FP
MIH, ALEXANOER, 1002 WEST 4TH,66720
473-2227
22 M 24209 47 ANES

CHAPMAN—913
(Dickinson County Society)

SVOBODA, CHARLES R, 413 N MARSHALL,67431
922-6400
18 M 1902 46 FP

CHERRYVALE—316
(Labette County Society)

MEKIS, JOHN, CHERRYVALE CLINIC,67335
336-2134
27 M 47302 53 FP

CHETOPA—316
(Labette County Society)

PEFFLY, ELMER O, GUY SHIELDS BUILDING,67336
236-7188
22 M 3901 53 FP

CLAY CENTER—913
(Clay County Society)

ANDERSON, SEVERT A, 532 LINCOLN,67432
632-3166
07 M 1902 36 00
CARLETON, RICHARD C, 709 LIBERTY,67432
632-5603
31 M 3005 61 FP
MCILVAIN, GUY B, 1601 THIRD,67432
—
97 M 1643 25 00
MCVAY, R BRUCE, 1503 FIFTH ST,67432
—
97 M 1902 29 00
OOONNELL, RICHARD H, 709 LIBERTY,67432
632-3101
16 M 1902 41 GS
RICHAROS, DENNIS O, 115 S 6TH,67432
632-5621
34 M 1902 60 FP

CLYDE—913
(Cloud County Society)

FREEBORN JR, WARREN S, ,66938
446-2221
26 M 1720 51 FP

COFFEYVILLE—316
(Southeast Kansas Society)

BRYANT, HOMER L, 803 W 9TH,67337
—
99 M 3501 30 00
CAMPBELL, WILLIAM H, 1411 W 4TH,67337
251-3235
39 M 1902 65 OPH
COYLE, JOHN F, 209 W 7TH,67337
251-2400
21 M 1902 44 FP
OICKINSON, CHARLES R, 108 W 7TH,67337
251-1340
20 M 1606 44 GS
EDROZO, M LUZ, MEMORIAL HOSPITAL,67337
—
F 74801 68 PATH
ELLIS, STEPHEN S, 1411 W 4TH,67337
251-3360
11 M 2802 36 S
GIBBS, EUGENE, 808 WILLOW,67337
—
M 64914 68 FP
HA, SANG W, 504 WILSHIRE,67337
251-7750
35 M 58309 60 OBG
HAN, CHAN S, 908 SIGGINS,67337
251-1560
35 M 58306 61 PD
HOWERTER JR, BERNARO E, BOX 659,67337
251-4790
43 M 1803 68 U
MARR JR, JAMES C, PO BOX 398,67337
251-1880
21 M 2834 45 ORS
MARTIN JR, ALBERT E, PO BOX 728,67337
351-2350
11 M 1902 37 FP
READ, WILLIAM T, 1411 WEST 4TH,67337
251-1120
16 M 2802 40 FP
SANOHU, PAUL S, PO BOX 257,67337
251-2450
42 M 49508 65 GS
TYLER, WILLIAM F, 808 WILLOW,67337
251-2100
42 M 1902 68 ORS
UY, WILSON O, COFFEYVILLE MEM HOSPITAL,67337
251-1200
42 M 74801 67 PATH
VAKAS, JOHN L, 1508 W 4TH,67337
251-3443
38 M 1902 64 IM
WHITE, OONALD C, COFFEYVILLE MEM HOSPITAL,67337
251-1200
35 M 3515 65 R

COLBY—913
(Northwest Kansas Society)

DAHL, ASHER W, COLBY CLINIC,67701
462-3333
28 M 1902 58 FP
DUNN, DANIEL R, COLBY CLINIC P O BX 28,67701
—
M 1902 74 FP
HASSETT, GERARO R, 1875 HARVEY CT,67701
—
HILOYARO, VICTOR H, COLBY CLINIC,67701
462-3333
16 M 1902 42 OTO

HILDYARD II, VICTOR H, BOX 28,67701
462-3332
47 M 702 73 FP
MARSHALL, GEORGE O, COLBY CLINIC,67701
462-3333
09 M 1902 36 FP
REGIER, LADONNA M, COLBY CLINIC,67701
462-3332
47 F 1902 73 FP
SMITH JR, FLOYD L, COLBY CLINIC,67701
462-3333
20 M 1902 44 FP

COLDWATER—316
(Iroquis County Society)

CHOUDHURY, M HASAN, 139 E MAIN ST PO BOX 65,67029
582-2136
46 M 70410 69 GS

COLUMBUS—316
(Cherokee County Society)

BELCHER, GEORGE O, COLUMBUS CLINIC,66725
429-2557
34 M 1902 60 FP
JONES, FORREST H, 219 S KANSAS,66725
429-3744
25 M 1902 54 FP
PASIMIO, ROGER S, RR #2,66725
429-1977
38 M 74801 62 GS

COLWICH—316
(Sedgwick County Society)

LIES, BARTHEL N, ,67030
796-1255
11 M 2802 37 FP

CONCORDIA—913
(Cloud County Society)

BRAY, AVIS PAGE, 1010 3RD AVE,66901
243-1560
17 F 702 54 FP
BUTT, MUHAMMAD, CONCORDIA MED GROUP,66901
243-1560
46 M 70401 69 GS
COULTER, THAYNE A, 11TH & WASHINGTON STS,66901
243-4455
12 M 2878 37 GP
FOWLER, WAYNE L, CONCORDIA MED GROUP,66901
423-1560
23 M 1720 47 IM
GELVIN, E RAYMOND, 835 WEST 9TH,66901
03 M 3005 27 OO
HOFER, DEWAYNE O, COUNTRY CLUB RD,66901
243-1263
36 M 1606 62 R
KOSAR, CLARENCE O, BOX 362,66901
98 M 1902 26 OO
LAWTON, MARVIN K, CONCORDIA MED GROUP,66901
243-1560
31 M 3005 58 GS
LLOYD, JAMES W, 810 WEST 11TH,66901
243-7011
44 M 1902 73 FP
MCCOMAS JR, MARMADUKE O, 11TH & WASHINGTON ST,66901
243-2511
16 M 1902 43 U
MOORE, JAMES E, 520-B WASHINGTON,66901
243-1094
48 M 1902 74 P
NELSON, PAUL L, 1010 THIRD AVE,66901
243-1560
27 M 1902 55 PD
NIXON, RICHARD R, 519 WASHINGTON,66901
243-1263
32 M 1643 57 R
OWENSBY, LINDELL C, ST JOSEPH HOSPITAL,66901
243-3386
21 M 2802 44 OPH
RUZICKA, LAWRENCE J, 1010 3RD AVE,66901
243-1560
13 M 3005 40 ANES
STRYKER JR, HENRY B, 717 FIRST AVE,66901
19 M 3501 44 IM
THORNTON JR, FOXHALL P, 1010 3RD AVE,66901
243-1560
25 M 5101 50 IM

COTTONWOOD FALLS—316
(Flint Hills Society)

MCKEE, LEO F, ,66845
273-6681
16 M 1902 39 FP

COUNCIL GROVE—316
(Flint Hills Society)

BARKER, ROYAL A, 221 HOCKADAY,66846
767-5126
21 M 1902 53 FP
BLACKBURN, ROBERT W, 221 HOCKADAY,66846
767-5126
22 M 1902 49 FP
SCHULTZ, JAMES E, 221 HOCKADAY,66846
767-5126
26 M 1902 56 FP

CUNNINGHAM—316
(Wyandotte Society)

ALLBRITTEN JR, FRANK F, PO BOX 177,67035
14 M 4101 38 OO

DENTON—913
(Northeast Kansas Society)

YODER, EMERSON O, ,66017
359-6531
14 M 1902 49 FP

DERBY—316
(Sedgwick County Society)

MCKERRACHER, ROBERT O, 400 A NORTH BALTIMORE,67037
688-1779
27 M 3901 55 FP
MOYER, HERMAN J, 200 S BALTIMORE,67037
788-1484
24 M 3901 55 FP

DIGHTON—316
(Southwest Kansas Society)

VON LEONROD JR, GEORGE, PO BOX 146,67839
397-5314
16 M 1902 43 FP

DODGE CITY—316
(Ford County Society)

AMAWI, MOHAMMAD S, DODGE CITY MED CENTER,67801
225-1371
46 M 87501 71 GS
AVILA, OSCAR A, DODGE CITY MED CENTER,67801
225-1371
41 M 17603 69 IM
BAUM, ARNOLD H, 2020 CENTRAL,67801
225-1371
16 M 2107 44 OBG
BOLES, R DALE, 2020 CENTRAL,67801
225-1371
23 M 3901 53 PD
BROWNRIGG, RICHARD L, 2020 CENTRAL,67801
225-1371
35 M 1902 61 U
BUSCH, ANTHONY B, 806 2ND AVENUE,67801
227-3141
05 M 2002 31 OO
CHOTIMONGKOL, ANUPONG, DODGE CITY MED CENTER,67801
225-1371
43 M 89102 69 OBG
CONARD, CLAIR C, DODGE CITY MED CENTER,67801
225-1371
27 M 1902 55 IM
JAMBOR, JAMES J, 304 HWY 56 WEST,67801
227-3213
20 M 2307 46 O

JOHNSON, HOWELL O. 2020 CENTRAL,67801
225-1371
45 M 1902 71 IM
*KUNNAWUTHIDEE, KAMOLTIPYA. 2020 CENTRAL,67801
-

MCCOY, RONALD. 806 SECDND AVE,67801
227-3141
19 M 3901 47 FP
MCELHINNEY, CHARLES F. 2020 CENTRAL,67801
227-1371
36 M 1902 62 GS
MIMOSO, JOSE J. 806 2ND AVE,67801
227-3141
37 M 4201 61 OBG
OHMAN, RICHARD J. 714 2ND,67801
227-2106
15 M 2407 41 S
REDDY, SATTI S. 2020 CENTRAL AVE,67801
225-1371
35 M 49504 66 U
SCHWARTZ, EUGENE W. 1ST NATL BANK BLDG,67801
225-4261
24 M 1902 50 OPH
STOCKWELL, MORGAN U. 2020 CENTRAL,67801
227-1371
24 M 1902 55 IM
TREKELL, WILLIAM V. SOUTHWEST CLINIC,67801
227-3141
25 M 1902 52 ORS
WILLIAMS, EVAN ROBERT. 2020 CENTRAL,67801
227-1371
25 M 1606 52 FP
ZACHARIAS, CARL KURT. 2020 CENTRAL,67801
227-1371
21 M 40707 47 ORS

DOUGLAS—316
(Sedgwick County Society)

MILLER, CLYDE W. RT 2 BOX 206 A,67039
-
09 M 2002 36 00

EL DORADO—316
(Butler-Greenwood Society)

BRIAN, ROBERT M. 123 N ATCHISON,67042
321-1230
02 M 1606 30 00
DELLETT, KENNETH B. 5TH & MAIN PLAZA,67042
321-1910
30 M 1902 55 OPTH
GIROD, CHARLES I. 123 N ATCHISON ST #103,67042
321-4981
11 M 4706 44 GS
HAFFNER, WILLIAM N. 123 N ATCHISON,67042
321-5049
35 M 1902 61 GS
HAMMEL, GEORGE W. 119 N JONES,67042
321-2010
08 M 1902 30 IM
JACOB, KANNAMPALLY L. 123 N ATCHISON,67042
321-0056
31 M 49537 59 U
JOHNSON, JOSEPH H. 119 N JONES BOX 1288,67042
321-3430
99 M 401 27 00
KASSEBAUM, GLEN E. 123 N ATCHISON,67042
321-2710
98 M 1606 23 00
LOWRY, RAY F. 226 WEST CENTRAL STREET,67042
321-9122
13 M 1902 45 FP
OLSEN, PHILLIP S. 123 N ATCHISON,67042
321-2100
46 M 1902 73 IM
OVERHOLSER, NORMAN H. 119 N JONES,67042
321-2010
16 M 1902 41 FP
PROCTOR, ROBERT W. 119 N JONES,67042
321-2010
38 M 1902 63 FP
SANDBERG, CHRIS B. 119 NORTH JONES,67042
321-2010
48 M 1902 74 FP
SHIELDS JR, JAMES M. 119 N JONES,67042
321-2010
18 M 4802 42 FP

WHITE II, BENJAMIN E. 119 N JONES,67042
321-2010
27 M 1902 54 FP
WILLIAMS, GENE V. 200 WEST ASH,67042
321-6680
23 M 1902 54 ENT

ELKHART—316
(Seward County Society)

PERIDO, DOMINADOR T. BOX 997,67950
697-2155
44 M 74801 68 GS
PULIDO, QUINTON M. 449 MORTON,67950
-
44 M 74801 68 GS

ELLINWOOD—316
(Barton County Society)

LAW, FINDLEY, MEDICAL ARTS BLDG,67526
564-2170
22 M 1902 51 FP

ELLIS—913
(Central Kansas Society)

ROCHANAYON, PIRA, 814 JEFFERSON ST,67637
726-4956
43 M 89101 69 FP
SURFACE, GARDNER A. 1204 WASHINGTON ST,67637
726-4212
02 M 1902 29 00

ELLSWORTH—913
(Central Kansas Society)

DAVIS, GEORGE R. 308 KINGSLEY,67439
472-3121
19 M 1902 44 GS
DDONNELL, H ST CLAIR, 308 KINGSLEY,67439
472-3121
93 M 2802 17 00
SEITZ JR, JOSEPH E. 308 KINGSLEY,67439
472-3121
22 M 1902 46 FP

EMPORIA—316
(Flint Hills Society)

BRADLEY, HOWARD R. 1601 STATE,66801
343-2900
35 M 1902 61 FP
BROCKHOUSE, JOHN P. 1601 STATE,66801
343-2900
31 M 1902 57 IM
BURGESON, FRANK G. 1601 STATE,66801
342-6989
40 M 3005 65 OPH
BUTCHER, THOMAS P. 1128 LAWRENCE,66801
342-0722
05 M 1601 34 00
CAMPBELL, EDWARD G. 1601 STATE,66801
343-2900
31 M 1902 61 FP
COLOSMITH, DONALD C. 1024 W 12TH,66801
342-7047
26 M 1902 58 FP
CONARD, RICHARD F. NEWMAN HOSPITAL,66801
342-7722
16 M 1902 42 R
DAVIS, DAVID R. 1038 LAWRENCE,66801
342-0646
02 M 2101 28 00
EDWARDS, DAVID J. 1601 STATE ST,66801
343-1191
43 M 2803 69 ORS
FRAZIER, RICHARD L. 1601 STATE ST,66801
343-2900
40 M 1902 72 GS
FUNES, CARLOS A. FLINT HILLS MED BLDG,66801
343-2900
42 M 13205 66 U
GANN, E LAMONTE, RR #2,66801
-
07 M 2802 37 00

GARCIA, GOULD C, 919 WEST 12TH AVE,66801
342-2521
32 M 3607 58 1M
GEITZ, JAMES M, 919 W 12TH,66801
342-2521
46 M 1902 72 1M
GINAVAN, DUANE A, 1024 W 12TH,66801
342-5876
35 M 1902 62 FP
GLENN, JAMES N, 1601 STATE,66801
343-1191
40 M 4804 66 ORS
HARVEY, JOHN E, 1601 STATE ST,66801
343-2900
39 M 1902 65 OBG
HAVENHILL II, MARSHALL A, 812 ANDERSON ST,66801
343-6400
35 M 1902 61 OBG
HOPPER, CHARLES R, 25 W 5TH,66801
342-2341
17 M 1902 47 FP
KNECHT, STEPHEN M, NEWMAN MEMORIAL HOSP,66801
342-7722
44 M 1902 70 R
LOHMEYER, KENNETH L, 1024 WEST 12TH,66801
342-6622
18 M 1902 44 FP
LUEDTKE, WALTER E, NEWMAN MEM HOS,66801
342-7120
18 M 5605 43 PATH
MIGUELIND, OLIVER M, NEWMAN HOSP,66801
342-7120
35 M 74801 57 PATH
MORGAN, JOHN L, 919 WEST 12TH,66801
342-2521
15 M 4101 40 1M
NEIGHBOR, RALPH M, 827 COMMERCIAL ST,66801
343-6565
46 M 1902 72 OBG
PARKER, WAYNE G R, STUDENT HEALTH CENTER,66801
343-1200
27 M 1902 56 FP
RYAN, EDWARD J, 919 WEST 12TH,66801
342-2521
12 M 1902 36 IM
RYAN, W SCOTT, 1601 STATE ST,66801
343-2900
47 M 1902 73 PO
SAUL, F WILLIAM, NEWMAN MEMORIAL HOSPITAL
342-7120
07 M 4113 40 R
SCHELLINGER, RICHARD P, 1128 LAWRENCE,66801
342-0722
22 M 3005 49 GS
SNOWBARGER, MARVIN O, 1601 STATE STREET,66801
343-2900
29 M 1902 55 FP
STEINKRUGER, VERLYN W, STUDENT HEALTH CENTER,66801
343-1200
28 M 3005 53 FP
TRIMBLE SR, DAVID P, 517 MERCHANT STREET,66801
342-2572
04 M 1902 32 OO
UNDERWOOD, CHARLES C, 25 WEST 5TH,66801
342-2341
07 M 1902 32 IM
VANDER VELOE, STANLEY L, 1601 STATE,66801
343-2900
16 M 1902 43 GS
WRIGHT, KENDALL M, 1024 WEST 12TH,66801
343-2376
45 M 1902 71 FP

ERIE—316
(Southeast Kansas Society)

BRYAN, EMERY C, RT 2 BOX 171,66733
-
04 M 1902 32 OO
WEIR, WILLIAM C, 115 S MAIN,66733
244-3771
17 M 2501 42 FP

ESKRIDGE—913
(Flint Hills Society)

WALKER, WILLIAM H, 2ND & CEDAR,66423
449-2301
10 M 2401 38 IM

EUDORA—913
(Douglas County Society)

HOLLADAY, KENNETH R, 101 WEST 10TH,66025
542-2345
34 M 1902 58 FP

EUREKA—316
(Butler-Greenwood Society)

BASHAM, JOHN H, BOX 469 824 E 3RD,67045
583-5554
11 M 2802 36 FP
CISKEY, WILLIAM J, 1602 NORTH ELM,67045
583-7401
47 M 1902 72 FP

FORT SCOTT—316
(Bourbon County Society)

AKERS, GUY I, 710 W 8TH,66701
223-3100
20 M 1902 53 FP
ALDIS, HENRY, 710 W 8TH,66701
223-3100
13 M 1902 41 OBG
ALDIS, WILLIAM, 710 WEST 8TH,66701
223-3100
20 M 1902 44 GS
BASHAM, JAMES J, 102 S JUDSON,66701
223-4100
14 M 1902 37 FP
BENAGE, JOHN F, 710 W 8TH,66701
223-3100
32 M 1902 58 OBG
BRAUN, EDWARD W, 710 WEST 8TH,66701
223-3100
42 M 1902 68 U
BURKE, JAMES J, 710 W 8TH,66701
223-3100
35 M 2834 61 IM
CHOW, STANLEY Y, 821 BURKE,66701
223-2200
18 M 24222 39 R
DUNSHEE, CARLYLE M, 710 W 8TH,66701
223-3100
32 M 1902 57 GS
GETTLER, DEAN T, 710 WEST 8TH,66701
223-3100
31 M 1902 57 GS
GOOD, JAMES T, 821 BURKE,66701
223-2200
21 M 2802 45 PATH
IRBY, ADDISON C, 416 S JUDSON,66701
-
05 M 1606 28 OO
IRBY, PRATT, 710 WEST 8TH,66701
223-3100
13 M 4705 36 U
MCCANN, PATRICK E, 710 WEST 8TH,66701
223-3100
28 M 1902 59 IM
MCKENNA, MICHAEL J, 102 SOUTH JUDSON,66701
223-4100
38 M 1902 64 FP
NELSON, T EUGENE, 710 W 8TH,66701
223-3100
41 M 1902 69 FP
REEVES, CHARLES S, 710 W 8TH,66701
223-3100
37 M 1902 63 IM
WEDDLE, DOUGLAS P, 710 WEST 8TH,66701
223-3100
43 M 1720 69 FP

FREDONIA—316
(Southeast Kansas Society)

BAYLES, HUGH G, 1508 MADISON,66736
378-3412
25 M 1902 52 FP

BEAL, RAYMOND J, 600 MADISON,66736
378-2159
12 M 1902 38 GS
HAYDEN, ABIGAIL, 617 MONROE STREET,66736
-
88 F 1902 35 OO
SUMNER, RALPH N, 712 MADISON,66736
378-2311
31 1902 57 FP

GARDEN CITY—316
(Southwest Kansas Society)

ARTILES, BENJAMIN H, 608 N 5TH, 67846
275-6111
29 M 54 P
AUSTIN, JOHN O, 601 N 6TH,67846
276-2346
14 M 1601 40 FP
BARNARD III, JAMES A, 1601 HARDING, 67846
275-9671
31 M 4812 62 OBG
BEGGS, DAVID F, 609 N STH,67846
276-9211
39 M 1902 64 IM
BIGLER, F CALVIN, GARDEN MED SPECIALISTS,67846
275-9671
31 M 801 57 GS
BRUNO, JAMES W, 1133 KANSAS PLAZA,67846
276-8201
42 M 4706 66 FP
EICHHORN, FRANK O, BOX 719,67846
276-8132
25 M 1902 56 FP
FRY, LUTHER L, 603 N 5TH,67846
27S-9671
41 M 1902 67 OPH
GILBERT II, JOHN H, 601 N 5TH,67846
275-9671
46 M 1902 70 ORS
GREENBERG, GEORGE E, ST CATHERINE HOSPITAL,67846
276-8241
42 M 401 68 R
HEARD, KENNETH M, 1109 PERSHING,67846
275-6111
21 M 4706 44 PATH
HERNANDEZ, ALEXANDER M, ST CATHERINE HOSP,67846
276-8141
33 M 84704 S9 P
JENKINS, EDWARD, 1133 KANSAS PLAZA,67846
276-8201
M 56 FP
KALBAC, RICHARD W, 603 N FIFTH,67846
275-9671
45 M 2803 70 OBG
MAXFIELD, RUSSELL J, 1133 KANSAS PLAZA,67846
276-8201
16 M 1902 41 FP
MEYERS, STEPHEN, 603 N 5TH, 67846
-
M 2834 PD
MILLER, ROBERT E, 603 N 5TH,67846
27S-9671
26 M 4812 55 GS
PATTERSON, MICHAEL S, 603 N 5TH,67846
27S-9671
43 M 1902 69 PM
SPIKES, MARION E, 603 N 5TH,67846
275-9671
26 M 1902 62 FP
TEARE, MAX E, 1007 DAVIS,67846
276-7689
28 M 1902 54 P
TURNER, JOHN W, 210 E SPRUCE,67846
276-3292
13 M 1902 39 FP
WILEY, HORACE M, 602 NORTH 6TH,67846
376-6901
12 M 2802 40 GS
ZELLER, MYRON J, 609 N 5TH,67846
275-9671
38 M 1902 64 OM

GARDEN PLAIN—316
(Sedgwick County Society)

BIERMANN, ALOYSIUS H, ,67050
535-2231
99 M 3006 23 OO

REINHARDT-WULF, TAISSIA L, PO BOX 273,67050
-
19 F 91302 42 OO

GARDNER—913
(Johnson County Society)

PRIETO, JORGE N, 427 W MAIN,66030
884-8711
4S M 26401 69 GS
REECE, A THOMEN, 121 HICKORY CIRCLE,66030
884-8711
37 M 1902 63 FP
REECE, ADELBERT S, 237 W MAIN,66030
-
01 M 1902 29 OO
REECE, CAROL A, 121 HICKORY CIRCLE,66030
884-8711
37 F 1902 63 PD

GARNETT—913
(Anderson County Society)

DOUGHERTY, THOMAS M, 117 W 6TH,66032
448-5421
28 M 1902 S5 FP
HARRIS JR, CLAIB B, 320 S OAK ST,66032
448-5431
17 M 1902 44 FP
LEITCH, DAVID A, GARNETT MEDICAL CENTER,66032
448-S421
38 M 1902 63 FP
STEVENS, MILDRED J, 202 W 4TH,66032
448-54S4
23 F 1902 47 FP
STEVENS, ROBERT L, 202 WEST 4TH,66032
448-5454
23 M 1902 47 FP

GIRARD—316
(Crawford County Society)

FRIGGERI, ROBERT W, 111 N SUMMIT,66743
724-8723
23 M 1902 51 FP
HALL, WESLEY H, 606 W ST JOHN,66743
724-6154
25 M 1902 57 FP

GLASCO—913
(Cloud County Society)

HARWOOD, CLAUDE J, PO BOX 428,67445
568-2342
25 M 1902 SS FP

GOESSEL—316
(Marion County Society)

CANELLOS, JAMES, GOESSEL CLINIC,67053
367-2205
30 M 86902 62 FP

GOODLAND—913
(Northwest Kansas Society)

AUSTIN, KENNETH O, 520 MAIN,67735
899-3633
33 M 3005 63 FP
BARE, LAWRENCE E, 520 MAIN,6773S
899-S651
37 M 3901 62 FP
LONG, LLOYD O, 520 MAIN,67735
899-S651
37 M 1720 63 FP
OLSON, CLITUS W, 520 MAIN ST,67735
899-3633
16 M 3005 48 GS
RENNER, MARION J, 126 NORTH 10TH,6773S
-
96 M 1902 22 OO

GREAT BEND—316
(Barton County Society)

ANDERSON, LYLE B, PO BOX 1285,67530
793-8141
28 M 1902 60 FP

BEAHM, ANDL W. 3923 BROADWAY,67530
793-7827
16 M 1902 43 FP
BEAHM, DONALD E. MED ARTS BLDG,67530
792-3626
45 M 1902 71 OPH
BROWN, C REIFF, 1031 JACKSON,67530
792-4391
31 M 3901 57 ORS
CAVANAUGH, CLAIR J. ST ROSE HOSPITAL,67530
792-2617
23 M 1803 47 R
DEGNER, JAMES B. 3515 BROADWAY,67530
792-2617
31 M 1902 57 R
ELLEDGE, E FRED, 1017 C JACKSON,67530
792-3678
38 M 1902 65 U
EVANS, WILLIAM R. 3923 BROADWAY,67530
793-3574
25 M 1902 53 FP
FIESER, CARL W. 3515 BROADWAY,67530
792-2617
45 M 1902 71 R
GATENO, JOSEPH, 3421 FOREST,67530
793-3501
25 M 64901 50 OBG
HOLT, JOHN M. 2200 LAKIN,67530
793-8429
35 M 1902 61 IM
JONES, EDWARD L. 3515 BROADWAY,67530
792-2511
35 M 1902 61 PATH
KING, WILLIAM T, 3431 FOREST,67530
793-3501
35 M 1902 61 OBG
KIRBY, MERLIN G. 1012 WASHINGTON,67530
793-3091
31 M 1902 56 GS
KRUEGER, HAVEN C. 1034 JACKSON,67530
792-2163
32 M 1902 61 POH
MCALLASTER, WENDALE E. 2111 FOREST,67530
793-3591
24 M 1902 54 GS
NIEDEREE, W CURTIS, 1012 WASHINGTON,67530
793-3091
30 M 3006 56 GS
POLSON, ROBERT C. BOX A 1422 POLK ST,67530
793-8414
17 M 1902 42 OPH
*PRESTON, RICHARD, 2200 LAKIN,67530
793-8426
42 M 1902 69 IM
REPLOGLE, CHARLES B. 2111 FOREST,67530
793-3591
27 M 1902 53 FP
RUIZ, CARLOS M. 3107 TWELFTH,67530
792-3210
25 M 27501 52 P
SAYLER, JEROME, CENTRAL KS MEDICAL CENTER,67530
792-2511
20 M 4113 50 PATH
SCHUETZ, PERRY N. 1422 POLK BOX A,67530
45 M 1902 71 OPH
SHIVEL, DAVID G. 3523 FOREST,67530
793-3523
28 M 1902 55 FP
SWAN, MAJOR MARTIN. 3923 BROADWAY,67530
792-4540
06 M 1902 43 IM
UNREIN, ROBERT J. 1017A JACKSON,67530
792-2504
29 M 1902 58 FP
WHITE, CHARLES L. 2412 OCVE TERR RT 4,67530
06 M 1902 36 OO
WIGGS, JAMES W. 1027 JACKSON,67530
792-1336
36 M 1720 63 N

GREENSBURG—316
(Iroquois County Society)

BRADLEY, J RODERICK, 502 SOUTH WALNUT,67054
723-2127
23 M 1902 47 FP
WALDORF JR, MELVIN H. BRADLEY-WALDORF CLINIC,67054
723-2127
23 M 1902 47 FP

HALSTEAD—316
(Harvey County Society)

AILLON, ALEJANDRO J. HERTZLER CLINIC,67056
835-2241
39 M 26402 63 TS
BAILEY, COLIN, HERTZLER CLINIC,67056
835-2241
33 M 35205 59 GYN
BEUGELSDIJK, HENRY PETER, HERTZLER CLINIC,67056
49 M 1902 74 AN
BOUDREAUX, VELTIN J. HERTZLER CLINIC,67056
835-2241
37 M 4812 64 R
BURNETT, A DEAN, HERTZLER CLINIC,67056
835-2241
21 M 1902 52 GS
DECKER, DONALD D, HERTZLER CLINIC,67056
835-2241
31 M 1902 56 CO
GNAU, FREDERIC B, HERTZLER CLINIC,67056
835-2241
42 M 1902 68 OTO
GRADINGER, BILLENS C, HERTZLER CLINIC,67056
835-2241
06 M 1902 34 IM
HARMS, WILMER A, THE HERTZLER CLINIC,67056
835-2241
22 M 1902 56 OPH
HOLT, ROBERT G, 327 CHESTNUT,67056
835-2241
22 M 5104 44 OPH
HOOFER, WILFORD D, HERTZLER CLINIC,67056
835-2241
30 M 1902 55 TS
KOENEKE, IRENE A, 327 CHESTNUT ST,67056
835-2241
98 F 2802 27 GYN
MALONE, EUGENE M, HERTZLER CLINIC,67056
835-2241
23 M 1902 56 IM
MONTGOMERY, DAN, HERTZLER CLINIC,67056
MONTGOMERY-SHRT, RUTH GERTRUDE, HERTZLER CLINIC,67056
835-2241
10 F 1902 37 ENT
MORTON, JOHN E, HERTZLER CLINIC,67056
835-2241
99 M 35211 26 PSY
O GRADY, JOSEPH A, HERTZLER CLINIC,67056
835-2241
16 M 3545 41 IM
POKORNY, CHARLES, HERTZLER CLINIC,67056
835-2241
08 M 2834 34 PUD
PRENTISS, HAROLD, 4TH AND CHESTNUT,67056
835-2241
36 M 1720 62 RAD
RATE, ROBERT G, HERTZLER CLINIC,67056
835-2241
14 M 1803 39 TS
RIZZA, ROBERT G, RT #2,67056
30 M 1201 56 PD
ROACH, NEIL E, HERTZLER CLINIC,67056
835-2241
38 M 1902 67 CP
SHAH, SHARFUDDIN, HERTZLER CLINIC,67056
835-2241
31 M 70401 58 IM
STOFFER, ROBERT P, HERTZLER CLINIC,67056
835-2241
26 M 1902 48 IM
TAN, POLY, HERTZLER CLINIC,67056
835-3241
44 M 3901 69 P
TEJANO, NEONILO A, HERTZLER CLINIC,67056
835-2241
43 M 74808 67 ORS
VRANEY, GEORGE, HERTZLER CLINIC,67056
835-2241
43 M 5606 69 IM

WELCH, JACK W. HERTZLER CLINIC,67056
835-2241
18 M 1902 51 GS

HANOVER—913
(Northeast Kansas Society)

WARREN, ROGER D. BOX 38,66945
337-2214
31 M 1902 57 GS

HARPER—316
(Tri-County Society)

BELLAR, RALPH E, 1019 CENTRAL, 67058
896-3131
31 M 3005 60 FP

HAVEN—316
(Reno County Society)

HAINES, CHESTER W. 102 SUNSET LANE,67543
—
01 M 1606 28 00

HAYS—913
(Central Kansas Society)

ALLEN, JAMES E. NORTHRIDGE PLAZA #6,67601
628-3261
46 M 1902 72 IM
APPLEGATE JR, FRANCIS R. 1010 DOWNING,67601
628-8218
30 M 1902 55 OPH
ARTMAN, JOHN C. 200S LINCOLN DRIVE,67601
625-2518
24 M 2002 48 ANES
BULA, RALPH E. 2707 WALNUT,67601
625-2551
12 M 1902 37 00
CARLSON, EARL V. 2818 N VINE ST,67601
628-8221
31 M 300S 56 ORS
CECIL III, JOHN, BOX 833,67601
625-6521
43 M 4804 69 R
CHAVALITHAMRONG, HANSA, 10S W 13TH,67601
625-25S1
45 M 89101 69 OBG
CODY, DOROTHY, 2704 WOODROW CT,67601
628-4000
29 F 3607 53 P
CODY, JOHN, 2704 WOODROW CT,67601
625-8251
25 M 401 60 P
COX, ROBERT H, 2507 CANTERBURY ,67601
628-3051
43 M 1902 70 PD
CRAMM, RUSSELL E, 2003 MAIN,67601
625-2518
30 M 1902 56 GS
EDDY, MURRAY C, 2001 LINCOLN DR,67601
—
98 M 1601 26 00
EDDY, VICTOR M, 10S W 13TH,67601
625-2S51
29 M 1902 55 GS
ENBERG, ROBERT, 2507 CANTERBURY ,67601
628-3051
43 M 1902 69 PD
FLANDERS, H ALDEN, 201 EAST 7TH,67601
628-8251
23 M 1902 44 IM
HAIGLER, JAMES P, 234 WEST 11TH,67601
625-2537
13 M 3006 39 FP
HALLING, L WILLIAM, 1300 EAST 13TH,67601
625-5646
27 M 5002 57 PATH
HULL, NORMAN E, 111 W 10TH,67601
625-2518
24 M 1902 52 FP
HUTCHISON, GLEN C, 3200 COUNTRY LANE,67601
628-8251
21 M 1902 50 ANES
KANE JR, WILLIAM M, 2805 CANTERBURY ,67601
628-3245
27 M 1001 54 OBG

KIFER, C JAMES, BOX 833,67601
—
IM
LASLEY, MICHAEL B, 2707 VINE SUITE 7,67601
628-3217
45 1902 71 GS
MATTICK, IRVIN H, 2818 N VINE,67601
628-8221
18 M 2802 43 ORS
MEDINA, ANIBAL, 2707 VINE,67601
628-1019
34 M 4S101 62 U
MYRICK, MICKEY, 111 WEST 10TH,67601
—
NEIL, ROY NEWTON, PO BOX 1019,67601
628-3215
38 M 3005 65 PATH
NEWCOMB, WARD A, 1300 E 13TH, 67601
625-5646
47 M 3005 71 PATH
REYNOLDS, JEFFREY C, 412 FIRST NATL BANK,67601
625-7311
39 M 1902 64 ENT
REYNOLDS, LLOYD W, 2110 ASH,67601
—
10 M 3840 34 00
SILER, EUGENE T, 1010 DOWNING AVENUE,67601
628-8218
24 M 1902 52 OPTH
STUMP, HARL G, 3208 WILLOW ST,67601
628-2826
39 M 1902 6S GS
THAOA, NARONGSAK K, 3104 OLYMPIC LANE,67601
62S-25S1
44 M 89101 69 IM
TOVAR, MANUEL H, 102 W 25TH,67601
625-3457
39 M 26404 67 FP
WATTS, HARRY E, 1010 DOWNING AVE,67601
628-8218
27 702 54 OPTH
WEBER, WALLACE N, 2707 VINE SUITE 10,67601
628-3231
43 M 1902 69 D
WIEGMAN, HUGH ALAN, BOX 833, 67601
628-1808
34 M 1803 60 OPH
WILCOX JR, HOWARD L, PO DRAWER 430,67601
628-8221
44 M 1902 70 ORS

HAYSVILLE—316
(Sedgwick County Society)

MORGAN, NOVA L, 301 W GRAND,67060
524-3275
20 M 3901 50 FP

HERINGTON—913
(Dickinson County Society)

OOZIER, FRED S, 1005 NORTH 8 STREET,67449
258-2215
10 M 4804 34 FP

HESSTON—316
(Harvey County Society)

DIENER, CLAYTON H, PO BOX 386,67062
327-4122
18 M 1902 54 GS
FRIESEN, FLORENCE V, SHOWALTER VILLA,67062
—
87 F 1611 14 00
YODER, VERNON E, ROUTE #1,67062
283-2400
31 M 4802 61 P

HIAWATHA—913
(Northeast Kansas Society)

ARANA, VICTOR, 107 S 6TH,66434
—
M 73701 61 GS
BUCKETT, THOMAS G, 314 OREGON,66434
742-2161
10 M 1902 34 FP

LARSON, DELBERT L, 314 OREGON,66434
742-2161
30 M 1803 64 FP
LOWE, DEWITT S, 202 S 6TH,66434
742-2117
99 M 3006 28 FP
MEIDINGER, RAY, 120 N 6TH,66434
742-2135
03 M 3005 32 00
SINNING, GARY, 314 OREGON,66434
-
M 1902 74 FP
WALTON, PHILIP D, 314 OREGON ST,66434
626-3322
32 M 1902 63 FP

HIGHLAND—913
(Northeast Kansas Society)

CORDER, ROBERT L, ,66035
442-3341
23 M 1902 49 FP

HILL CITY—913
(Northwest Kansas Society)

REDDY, B N, 304 WEST PROUT,67642
674-2255
38 M 49557 66 PD
REDDY, P JAGANNADHA, 80 WALNUT DRIVE,67642
674-2255
42 M 49511 66 GS

HILLSBORO—316
(Marion County Society)

ENS, GERHARD GEORGE, 613 SOUTH MAIN,67063
947-5931
20 M 1902 55 FP
ENS, PETER, 209 SOUTH MAIN, 67063
947-3671
14 M 1902 51 00
FRANZ, ROBERT G, 704 S MAIN,67063
947-3197
33 M 3901 59 FP
JANZEN, HERMAN F, 128 S MAIN,67063
-
91 M 1902 35 00

HOISINGTON—316
(Barton County Society)

MOORE, ROBERT, 814 NORTH ELM,67544
653-2151
22 M 3901 53 FP
WOLFENBARGER, KEITH A, 351 WEST 10TH,67544
653-4145
27 M 1902 58 FP

HOLTON—913
(Jackson County Society)

MOSER, ERNEST C, 438 HILLCREST DR,66436
-
09 M 1902 34 00
MOSER, M ROSS, 418 WEST FIFTH,66436
364-3116
19 M 1902 47 FP
MOSER, ROY H, 801 IOWA,66436
-
04 M 1902 32 00
SEELEY, JAMES C, 418 WEST 5TH,66436
364-3116
34 M 1902 64 FP

HORTON—913
(Northeast Kansas Society)

WOOD, ROBERT D, 105 W 8TH,66439
486-2127
26 M 1902 53 FP

HOXIE—913
(Northwest Kansas Society)

NEUENSCHWANDER, JOHN, 700 MAIN,67740
675-3292
26 M 2802 51 FP
NEUENSCHWANDER, JOHN RAND, 700 MAIN,67740
675-3292
47 M 1902 72 FP

HUGOTON—316
(Seward County Society)

FREDERICK, MAC F, PO BOX 909,67951
544-2784
20 M 1902 44 FP
LENEVE, ROBERT T, PO BOX 909,67951
544-2784
21 M 3901 46 FP

HUMBOLDT—316
(Southeast Kansas Society)

LONG, EDWARD E, 8TH & NEW YORK,66748
473-2411
21 M 1902 50 FP

HUTCHINSON—316
(Reno County Society)

ADAMS JR, MARCUS W, 1125 N MAIN,67501
663-6121
33 M 3901 59 PD
ALBRIGHT, JEROLD D, 1125 N MAIN,67501
663-6121
39 M 1902 66 FP
ANDERSON, DAVID G, 1100 N MAIN,67501
669-0191
35 M 1602 61 ORS
ANTLFINGER, THOMAS J, HUTCHINSON HOSPITAL,67501
663-6811
42 M 5606 67 R
AUSTIN, GEORGE M, 1125 N MAIN,67501
663-6121
42 M 1720 68 U
BAKER, ROBERT B, HUTCHINSON HOSP,67501
663-6811
47 M 6001 72 R
BAUER, THOMAS A, HUTCHINSON CLINIC,67501
663-6121
41 M 1902 67 IM
BLANK, JOHN N, WOLCOTT BLDG,67501
663-4271
07 M 1902 38 FP
BORRA, MARIO J, 1 WEST 8TH,67501
662-1751
24 M 2401 47 U
BOS, NORMAN C, 2415 N MAIN,67501
663-4406
24 M 1611 47 ORS
BURGER, J DALE, 1125 N MAIN,67501
663-6121
21 M 1902 46 FP
CASADY, GILBERT N, 39 LINKSLAND DR,67501
663-3657
23 M 1902 53 ANES
CASEY, JAMES, 1125 NORTH MAIN,67501
-
M 3005 69
CHEN, JOHN S, 215 COUNTRYSIDE DRIVE,67501
662-4427
33 M 24216 56 ANES
CHERVEN, PHILIP L, 1100 N MAIN,67501
662-3364
45 M 2501 71 PD
CRAWFORD, ROBERT A, 1125 N MAIN,67501
663-6121
16 M 1601 42 FP
DEPENBUSCH, FRANCIS L, 512 WOLCOTT BLDG,67501
663-7187
38 M 1902 65 OPH
ECKART, DE MERLE E, 312 WOLCOTT BLDG,67501
662-4921
14 M 1902 40 P
FALTER, RICHARD T, 512 WOLCOTT BLDG,67501
663-7187
38 M 1902 67 OPH
FERNIE, ROBERT W, 1100 N MAIN,67501
663-1136
06 M 702 34 IM

FOSS, DANIEL C. 1125 N MAIN,67501
663-6121
43 M 1902 69 GE
GOODPASTURE, WILLARD C. WILLOWBROOK, HUTCHINSON, 67501
10 M 1602 36 OO
HEDRICK, KENNETH E. 1125 N MAIN,67501
663-6121
27 M 1902 53 GS
HILL, JOHN J. 519 WILEY BLDG,67501
662-1291
16 M 1902 44 OTO
HINSHAW JR, CHARLES T. 724 N MAIN, 67501
662-7802
32 M 1902 58 PATH
HOLDERMAN, WALLACE O. 2415 N MAIN,67501
663-4406
28 M 1902 54 ORS
JARROTT, JOHN B. 1100 N MAIN,67501
669-0191
16 M 1902 40 ORS
LETTNER, HANS T. 725 N MAIN,67501
662-7801
23 M 40716 49 PATH
LUKENS, DAVID. 1100 N MAIN,67501
663-1136
24 M 2307 48 IM
MCCOY, CHARLES T. 310 WOLCOTT BLDG,67501
662-0121
16 M 1902 41 OPH
MCMULLEN, JOSEPH E. 1125 N MAIN,67501
663-6121
33 M 1902 62 GS
MULL, JOHN C. 1125 N MAIN,67501
663-6121
34 M 1902 61 OBG
NEEL, WILBUR B. 1125 N MAIN,67501
663-6121
24 M 1902 59 IM
NEUSCHAFER, DARREL R. 1125 N MAIN,67501
663-6121
48 M 1902 74 OBG
NUNEMAKER, MARION E. 919 N MAIN,67501
662-5391
21 M 1902 46 ANES
OPENSHAW, CALVIN R. 1100 N MAIN,67501
662-3305
21 M 4901 44 GS
PAINE, GEORGE E. WILEY BLDG,67501
94 M 1606 19 OO
PERKINS, JACK L. 1125 N MAIN,67501
663-6121
24 M 1902 53 FP
SCHROLL, JACK C. 1125 N MAIN,67501
663-6121
24 M 1902 49 OBG
SHAW JR, JAMES W. 725 N MAIN,67501
662-7801
40 M 1902 65 PATH
SHEARS, ROBERT N. 1100 N MAIN,67501
662-3364
20 M 1902 44 PD
STAFFORD, ROBERT W. HUTCHINSON CLINIC,67501
663-6121
43 M 2101 69 IM
STENSAAS, CARL O. 401 WOLCOTT BLDG,67501
663-9147
10 M 1902 38 D
STOUT, JAMES M. 1125 N MAIN,67501
663-6121
29 M 1902 55 FP
SUMNER, JOYCE R. 2027 N MADISON,67501
663-0822
26 F 1902 51 ANES
SUMNER, MARION M. 1125 N MAIN,67501
663-6121
26 M 1902 52 IM
TAYLOR, ELWYN J. 1100 N MAIN,67501
663-1251
34 M 1902 61 FP
TWEITO, DAVID H. 1100 N MAIN,67501
662-3364
38 M 1803 64 PD
VON RUDEN, WILLIAM J. 1100 N MAIN,67501
662-3306
26 M 1611 52 GS
WOLFE, JOHN E. 1125 N MAIN,67501
663-6121
42 M 1902 68 IM
WORTMAN, JACK A. 1125 N MAIN,67501
663-6121
34 M 1902 62 IM

INDEPENDENCE—316
(Southeast Kansas Society)

BARBERA, PORTER E. 800 WEST CHESTNUT,67301
331-4400
19 M 4707 46 FP
BEAHM, EDGAR H. 307 PROFESSIONAL BLDG,67301
331-2311
13 M 1902 44 FP
BULLOCK, HAROLD O. PO BOX 746,67301
06 M 1902 33 OO
CHAPPUIE, WILLIAM G. 800 CHESTNUT WEST,67301
331-5440
24 M 1902 51 FP
EMMOT, WILLIAM W. 800 WEST CHESTNUT,67301
331-6350
45 M 1902 71 IM
EMPSON, CHARLES L. 400 N 14TH,67301
331-6019
37 M 1902 68 FP
GAREY, WILLIAM JOHN. MERCY HOSPITAL,67301
331-2200
44 M 6501 70 R
KNUTH, KENNETH L. 115 MORNINGSIDE PL, 67301
331-2200
22 M 1902 50 R
MYERS JR, EARL B. 800 WEST CHESTNUT,67301
331-3420
32 M 2803 64 GS
ROBINSON, EDGAR L. 209 NORTH 6TH,67301
331-1450
15 M 1902 42 FP
SHEIKH, MASOOD A. 900 W MYRTLE,67301
331-2803
44 M 70402 66 GS
STRAHM, WAYMER J. RT 1,67301
331-1748
29 M 1902 62 P

IOLA—316
(Allen County Society)

COPENING, TELL B. 826 E MADISON,66749
365-2131
43 M 1902 69 FP
DETAR, GEORGE F. 219 W MADISON,66749
365-3712
28 M 1902 57 GS
OICK, WILLIS G. IOLA MEDICAL ARTS BLDG,66749
365-2131
13 M 512 41 GS
HANSON, DAVID C. 826 E MADISON,66749
365-2131
46 M 512 73 FP
LENSKI JR, FRANCIS X. 206 S JEFFERSON,66749
365-3901
26 M 1606 49 FP
MYERS, W EUGENE. 211 S STREET,66749
365-3732
12 M 1902 46 FP
PEES, GERALD B. 219 W MADISON,66749
365-5175
15 M 1902 43 GS
SCHMAUS, LYLE F. 202 E STREET,66749
365-3611
99 M 1803 26 OO

JETMORE—316
(Ford County Society)

OSHEA, JAMES G. BOX 545,67854
357-8321
18 M 3901 48 FP

JEWELL—913
(Mitchell County Society)

PLOWMAN, CARL W. ,66949
428-3511
99 M 1606 26 OO

JUNCTION CITY—913
(Geary County Society)

BOLLMAN, CHARLES S, PO BOX 397,66441
762-4575
41 M 3901 66 GS

BRETHOUR, LESLIE J, PO BOX 49,66441
238-4151
13 M 3006 39 FP

BUNKER JR, HERBERT L, 1106 ST MARYS RD,66441
238-5131
20 M 1606 45 FP

COPELAND, GARY A, GEARY COMMUNITY HOSP,66441
762-2387
42 M 1902 68 R

LABHSETWAR, SNEHALATA A, MED CLINIC BLDG SUITE 101,66441
762-4147
39 F 49528 64 OBG

MACE, RONALD C, PO BOX 163,66441
762-4884
42 M 3901 74 FP

MINNICK, CHARLES V, 602 NORTH JEFFERSON,66441
762-2051
08 M 2105 35 FP

NEMMERS, DAVID J, 1106 ST MARYS RD,66441
34 M 1803 59 IM

ODONNELL, HARRY E, 1106 ST MARYS RD,66441
762-4947
14 M 4113 42 FP

SCOTT, ALEX, 507 WEST 6TH,66441
238-2518
23 M 5605 48 FP

SMILEY, WILLIAM A, 240 W 5TH,66441
85 M 1903 10 OO

WRIGHT, STANLEY E, 1106 ST MARYS RD,66441
762-4884
47 M 3901 74 FP

KANSAS CITY, KANSAS—913
(Wyandotte County Society)

ABDOU, NABIH I, KU MEDICAL CENTER,66103
588-6586
34 M 33002 58 A

ALEXANDER, CHARLES E, 600 NEBRASKA,66101
321-6670
43 M 401 70 OBG

ALEXANDER, CLYDE W, 1514 NORTH 5TH,66101
281-4380
96 M 4707 23 OO

ALFIE, SALOMON, 3500 RAINBOW #202,66103
432-3300
33 M 13201 57 CHP

ALGIE, WILLIAM H, 907 N 7TH,66101
281-2929
02 M 1902 27 OO

ALLEN, WILLIAM R, 9201 PARALLEL PARKWAY,66112
334-4110
22 M 1902 46 R

ALLEN JR, WILLIAM R, 3615 CAMBRIDGE,66103
46 M 1902

AMARE, MAMMO, KUMC,66103
588-6031
36 M 60501 61 IM

ARAKAWA, KASUMI, KU MEDICAL CENTER,66103
588-6670
26 M 57211 53 ANES

ARENAL, ANGELA C, BETHANY HOSPITAL,66102
362-6389
38 F 35204 60 ANES

ARVANITAKIS, CONSTANTINE, KUMC,66103
588-6990
39 M 41802 65 IM

ARVANITAKIS, SANDA, KUMC,66103
588-6323
39 F 41802 64 PD

ASHER, MARC A, KUMC,66103
588-6130
36 M 1902 62 ORS

ATKINS JR, FLOYD L, KUMC,66103
588-6015
43 M 5104 69 CD

AZARNOFF, DANIEL L, KU MEDICAL CENTER,66103
588-6026
26 M 1902 55 IM

BAKER, WILLIAM S, KUMC,66103
588-6489
47 M 702 73 P

BALSYS, RAYMOND, UNIV OF KANSAS,66103
588-6800
44 M 26405 70 R

BARNHORST, DONALD A, KUMC,66103
588-6107
37 M 2834 63 TS

BASS JR, LEWIS N, 1975 N 5TH,66101
321-2320
21 M 1902 45 PD

BATNITZKY, SOLOMON, KUMC 39TH AT RAINBOW,66103
40 M 83601 64 R

BECKER, LESLIE E, 600 NEBRASKA,66101
342-4010
23 M 1003 46 U

BERGIN, JAMES J, BETHANY MED CENTER,66102
281-8767
28 M 2407 54 IM

BICHLMEIER, FRANKLIN G, 155 S 18TH,66102
371-6800
33 M 1902 58 TS

BIGONGIARI, LAWRENCE R, KU MED CENTER,66103
588-6000
44 M 1611 69 DR

BOGGAN, MICHAEL D, 155 S 18TH,66102
371-6800
40 M 1201 67 GS

BOLINGER, ROBERT E, KU MEDICAL CENTER,66103
588-6022
19 M 1902 43 IM

BOLTON, VICTOR E, 155 SOUTH 18TH,66102
371-2020
24 M 1902 48 OBG

BOSWELL, H CRAIG, KUMC 39TH AT RAINBOW,66103
588-6670
47 M 1803 73 ANES

BRACKETT JR, CHARLES E, KU MED CENTER,66103
588-6117
20 M 3501 44 NS

BRILLHART, MAXINE T, 1610 WASHINGTON BOULEVARD,66102
321-4800
15 F 1902 50 FP

BUAKHAM, CHANA, KUMC,66103
588-6670
37 89102 62 ANES

BURGER, WILLIAM E, 355 NEW BROTHERHOOD BLDG,66101
371-1017
21 M 3006 51 GS

BURKET JR, GEORGE E, DEPT OF FAMILY PRACTICE,66103
588-6907
12 M 1902 37 FP

CALKINS, W GRAHAM, KUMC 39TH AT RAINBOW,66103
861-4700
26 M 2501 50 GE

CAMERON, WILLIAM J, KU MEDICAL CENTER,66103
588-6200
29 M 2501 54 OBG

CARPENTER, PAUL R, 155 SOUTH 18TH,66102
371-6800
24 M 1902 50 TS

CARTER JR, ROBERT P, KUMC,66103
588-6217
40 M 3901 65 OBG

CASTEEL, CHARLES K, 155 SOUTH 18TH,66102
342-7233
34 M 3901 59 U

CAUGHRON, MICHAEL ROBERT, 5105 NEOSHO,66205
49 M 1902 74 PATH

CHALIAN, ALEXANDER R, 2648 MINNESOTA AVENUE,66102
588-5000
03 M 3509 37 OO

CHANG, CHAE H, K U M C,66103
588-6807
29 M 58301 53 R

CHIN, TOM D, DEPT OF HUMAN ECOLOGY,66103
588-7177
22 M 2501 43 IM

CHO, CHENG T. KUMC,66103
588-6336
37 M 38501 62 PD
CHONKO, ARNOLD M. KU MED CENTER,66103
588-6969
43 M 3840 69 NEP
CHRISTIAN, STANLEY J. 1003 CENTRAL AVE,66102
371-1181
18 M 1902 44 FP
CHUN, CHUNG S. KUMC,66103
588-6512
30 F 58301 53 PD
COALE, LLOYD H. 5020 GREELEY,66104
13 M 1902 43 OO
COOK, BRUCE A. KUMC,66103
49 M 1902
COOK, JAMES D. KU MED CENTER,66103
588-6031
36 M 6505 60 HEM
COOKE, ALLAN R. KUMC DEPT OF MED,66103
588-6990
36 M 14303 58 GE
COUCH JR, JAMES R. KUMC,66103
588-6970
39 M 4804 65 N
COULTER, THOMAS B. KUMC 39TH & RAINBOW,66103
931-0833
38 M 1205 64 OPH
COX III, IRA L. 155 SOUTH 18TH,66102
371-4343
43 M 1902 68 OR
COX JR, WALLACE F. 4631 ORVILLE,66102
321-1199
30 M 1902 56 GS
CRIST, ROBERT D. KUMC,66103
588-6200
36 M 1902 63 OBG
CROCKETT, CHARLES A. 155 S 18TH,66102
342-2200
19 M 401 44 OPH
CULP, LOUIS M. 1645 WASHINGTON BLVD,66102
371-1077
24 M 1902 53 FP
DAVIS, CHESTER R. 1100 COUNTY LINE RD B8A33,66103
588-6522
50 M 1902 75 FP
DAVIS, CHRISTOPHER G. 219 HURON BLDG,66101
321-9313
09 M 1902 39 FP
DAVIS JR, JAMES W. KUMC,66103
861-4700
33 M 4705 57 IM
DAY, HUGHES W. BETHANY MED CENTER,66102
281-8856
15 M 1902 39 IM
DEITZ, MICHAEL R. 155 S 18TH,66102
342-2222
32 M 4101 58 OPH
DIALLO, GASTON I. 600 NEBRASKA,66117
281-2888
35 M 86905 64 GE
DICK, ARTHUR R. KUMC,66103
588-6970
34 M 2301 65 N
DICKERSON, SHELBY C. BOX 700,66103
588-6015
46 M 5101 72 IM
DIEDERICH, DENNIS A. KUMC,66103
588-6981
36 M 2834 61 IM
DUDDING, BURTON A. KU MED CENTER,66103
588-6339
39 M 3520 64 PD
DUJOVNE, CARLOS A. KU MED CENTER,66103
588-7718
37 M 13201 61 CP
DUNN, MARVIN I. KU MED CENTER,66103
588-6015
27 M 1902 54 IM
EGEA, FERNANDO M. 6013 LEAVENWORTH,66104
334-3400
37 M 13206 62 N
FESTOFF, BARRY W. K U M C 39TH AT RAINBOW,66103
861-4700
40 M 1102 77 N
FITZPATRICK, M ROBERT, 1610 WASHINGTON BLVD,66102
321-4800
20 M 1803 44 FP

FLOERSCH, HUBERT M. 155 S 18TH,66102
371-2020
08 M 1902 35 OBG
FOROYCE, NORMAN. 255 N BROTHERHOOD BLDG,66101
321-1161
41 M 1902 67 ENT
FORET, JOHN O. KU MED CENTER,66103
588-6146
26 M 1602 53 U
FOSTER, FRANCES M. TWO GATEWAY CENTER,66101
621-1088
41 F 4707 67 FP
FOX, DEANNA K. KUMC 39TH AT RAINBOW,66103
588-6670
48 F 1902 74 ANES
FOX, HOWARD A. KUMC,66103
588-6337
33 M 3501 62 PD
FRANCISCO, CLARENCE L. 155 S 18TH,66102
371-6800
09 M 1902 34 OO
FRANCISCO, W DAVID. 155 S 18TH,66102
371-6800
21 M 1902 44 ORS
FRIESEN, STANLEY R. KU MED CENTER,66103
588-6108
18 M 1902 43 GS
FUNK, EDWARD O. BETHANY HOSP,
621-6600
04 M 1902 41 OO
GALLEHUGH, KEITH W. 155 S 18TH ST,66102
371-4343
32 M 1902 57 R
GILHOUSEN, FREDERIC M. 1029 N 32ND,66102
621-1066
40 M 1902 66 ORS
GILMORE, CLARENCE A. 600 NEBRASKA,66101
321-6670
31 M 4707 60 OBG
GODFREY, ROBERT G. KUMC,66103
588-5371
27 M 1902 58 RHU
GODFREY, WILLIAM A. KUMC,66103
588-6600
38 M 1902 65 OPH
GOERTZ, LEO R. 155 S 18TH ST,66101
371-4343
22 M 1902 52 R
GOODWIN, DONALD W. KUMC,66103
588-6402
31 M 1902 64 P
GOTO, HIROSHI, KUMC,66103
588-6670
42 M 57241 67 ANES
GRANTHAM, JARED J. KUMC,66103
588-6983
36 M 1902 62 IM
GREENBERGER, N J. KUMC,66103
588-6001
33 M 3806 59 IM
GRIPKEY, CLARENCE A. 707 NORTH 75TH,66112
05 M 1902 30 DO
GRUENDEL, RICHARD A. 1029 N 32ND,66102
281-5252
29 M 1902 55 ORS
GRUENDEL, VIRGINIA T. 6926 GARFIELD,66102
621-1066
30 F 1902 55 FP
GUMUCIO, MARIO L. 860 NEW BROTHERHOOD BLDG,66101
321-4046
30 M 64901 63 IM
GURWITH, MARC. KUMC 39TH AT RAINBOW,66103
588-6035
39 M 2401 65 IM
HAAS, CHARLES O. KUMC,66103
588-6029
39 M 1902 68 IM
HABERSANG, ROLF W O. KUMC,66103
588-6324
41 M 86901 70 PD
HANCOCK, ALAN C. 10111 LEAVENWORTH RD,66109
299-0130
35 M 1902 65 FP
HARA, GLENN S. 155 SOUTH 18TH,66102
588-6219
43 M 502 69 OBG
HARDIN, CREIGHTON A. KU MED CENTER,66103
588-6106
18 M 5605 43 GS

HARTMAN, CHARLES R, KUMC,66103
588-5551
37 M 1902 66 IM
HARTMAN, GERALD V, KUMC,66103
588-7350
20 M 1902 45 TR
HATHAWAY, PETER, KUMC,66103
588-6043
31 M 3503 60 IM
HERMRECK, ARLO S, KUMC,66103
588-7232
38 M 1902 65 GS
HIEBERT, PETER E, 155 S 18TH,66102
371-4343
03 M 1902 34 00
HINTHORN, DANIEL R, KUMC,66103
588-6035
41 M 1902 67 IM
HIRSCH, MARTIN S, KUMC,66103
588-6400
44 M 1902 71 P
HITCHCOCK, CHAS THOMAS, 155 SO 18TH SUITE 200,66102
371-2900
M 1902 78 GS
HODGES, GLENN R, KUMC,66103
588-6035
41 M 1602 67 IM
HOLDCRAFT, JACQUELYNE, 255 NEW BROTHERHOOD BLDG,66101
321-1161
36 F 2105 63 ENT
HOLLEMAN JR, JAMES F, D.O., 667 S 55TH ST,66106
287-1414
38 M 2878 68 FP
HOLLOWELL JR, JOSEPH G, KUMC,66103
588-7179
32 M 4501 59 PD
HOLMES, FREDERICK F, KUMC,66103
588-7175
32 M 5404 57 IM
HOLMES, GRACE E, KUMC,66103
588-6325
32 F 5404 57 PD
HOOGSTRATEN, BARTH, KUMC,66103
588-6029
24 M 66001 55 ONC
HORTON, WILLIAM A, KUMC 39TH AND RAINBOW,66103
588-6043
45 M 1902 71 IM
HUERTER, QUENTIN C, 771 NEW BROTHERHOOD BLDG,66112
321-5370
31 M 1902 59 OPH
HUFF, JOHN O, 1428 S 32ND,66106
384-1630
21 M 1902 52 FP
HUFFMAN, DAVID H, KUMC 39TH & RAINBOW,66103
861-4700
41 M 1902 67 IM
HUGHES, JOHN JAY, 155 S 18TH S 101,66102
281-8937
46 M 3509 71 DR
HUMPHREY, LOREN J, 70 TERR TRAIL,66106
588-6101
31 M 1611 56 GS
HURWITZ, ARYEH, KU MED CENTER,66103
588-7700
36 M 2802 61 IM
HUYCKE OUDGEON, M MAUREEN, 1912 W 37TH AVE,66103
-
51 F 1902 STU
IBARRA, RICHARD C, 754 PACIFIC,66101
342-3969
26 M 64902 57 FP
INGRAM, JOHN E, 1428 S 32ND,66106
384-1630
24 M 3006 56 FP
INNES, ROBERT C, 8929 PARALLEL PARKWAY,66112
334-2500
25 M 2802 49 R
JACKSON, ROGER P, KUMC,66103
588-6130
47 M 2803 73 ORS
JACOBS, DAVID S, 8929 PARALLEL PARKWAY,66112
334-2500
31 M 2501 56 PATH
JACOBS, RAE R, KUMC,66103
588-6131
36 M 3506 62 ORS
JAHANIAN, DARYOUSH, 155 S 18TH,66102
371-2020
40 M 51701 64 OBG

JAKOWATZ, JAMES G, 4308 RAINBOW,66103
-
53 M 1902 STU
JEWELL, WILLIAM R, KUMC,66103
588-6112
35 M 1611 61 GS
JOHNS JR, LEO F, KUMC 39TH & RAINBOW,66103
861-4700
22 M 2101 45 IM
JOHNSON, JOHN E, BETHANY MED CTR,66102
281-8816
17 M 4706 43 PATH
JONES JR, HERMAN H, 600 NEBRASKA,66101
342-4010
25 M 4707 54 GS
KALIVAS, JAMES T, KU MED CENTER,66103
588-6028
38 M 502 63 D
KAUFFMAN, RALPH E, KU MED CENTER,66103
588-6324
39 M 1902 65 PD
KENNEDY, JAMES A, KUMC,66103
588-6000
35 M 2834 61 IM
KEPES, JOHN J, DEPT OF PATH,66103
588-7076
28 M 47301 52 PATH
KERBY, GERALD R, KU MED CENTER,66103
588-6044
32 M 1902 58 PUD
KESTENBAUM, THELDA M, KUMC 39TH AT RAINBOW,66103
588-6028
48 F 5101 73 D
KETCHUM, LYNN D, KU MED CENTER,66103
588-6143
36 M 2101 60 PS
KIM, JONG M, KUMC,66103
588-6670
40 M 58302 64 ANES
KING, CHARLES R, KUMC, 66103
588-6200
47 M 1902 71 OBG
KIRCHNER, FERNANDO R, KUMC,66103
588-6700
30 M 64901 55 OTO
KIRSHNER, NANCY A, KUMC,66103
588-6800
44 F 4802 73 PDR
KRANTZ, KERMIT E, KU MED CENTER,66103
588-6201
23 M 1606 48 OBG
KRUEGER, ALAN L, KUMC, 66103
588-6412
41 M 1902 67 P
KWEE, SIOE T, 8929 PARALLEL PRKWAY,66112
334-2500
36 F 1720 63 PATH
KYNER, JOSEPH L, KU MED CENTER,66103
588-5554
34 M 1902 60 IM
LAING, ROBERT R, 155 S 18TH ST,66102
371-4301
37 M 1643 61 GE
LANSKY, LESTER L, KUMC,66103
588-6300
33 M 2604 65 PDN
LANSKY, SHIRLEY B, KUMC,66103
588-6302
35 F 2604 60 PD
LAYBOURNE JR, PAUL C, KU MED CENTER, 66103
588-6475
19 M 3509 44 P
LEAKE III, HUNTER C, KUMC,66103
588-6300
37 M 2105 63 PD
LEE, JAE M, 4631 ORVILLE SUITE 109,66102
677-3555
40 M 58302 65 GS
LEE, KYO R, KUMC,66103
588-6800
33 M 58302 59 R
LEE JR, JAMES G, 155 S 18TH,66102
371-2330
18 M 1902 44 OBG
LEITE, CAROL JO, KUMC 39TH AT RAINBOW,66103
588-6029
46 F 1902 72
LEMOINE JR, ALBERT N, KUMC,66103
588-6600
18 M 2802 43 OPH

LED, WILLIAM A, KUMC 39TH AT RAINBDW,66103
-
22 M 1902 48 GS
LEVINE, ERRDL, KUMC 39TH AT RAINBDW,66103
588-6800
41 M 83601 64 DR
LEWIS JR, H DANIEL, KUMC 39TH AT RAINBDW,66103
861-4700
32 M 5101 58 CD
LINDSLEY, CARDL B, KUMC,66103
588-6325
41 F 5404 68 PD
LINDSLEY, HERBERT B, KUMC,66103
588-6586
40 M 1902 66 RHU
LINSHAW, MICHAEL A, KUMC,66103
588-6323
40 M 4109 66 PD
LIPSCHITZ, DAVID A, KU MED CENTER,66103
831-6937
43 M 83601 66 HEM
LIU, CHIEN, KUMC 39TH AT RAINBDW,66103
421-6210
21 M 24217 47 PD
LLOYD, HARVEY L, 3200 STRONG AVE,66106
588-1111
08 M 1503 36 FP
LOWMAN, JAMES T, KUMC,66103
588-5701
31 M 401 58 PD
LUKERT, BARBARA P, KUMC 39TH AT RAINBDW,66103
588-5554
34 F 1902 60 END
LYNCH, SEAN R, KUMC 39TH AT RAINBOW,66103
588-6031
38 M 83601 61 IM
MACARTHUR, RICHARD IAN, KUMC 39TH AT RAINBDW,66103
588-6100
46 M 1902 73 GS
MAKLAD, M NABIL, KUMC
588-6884
38 M 33003 61 R
MANI, M MANI, KUMC,66103
588-6136
37 M 49527 60 PS
MANSFIELD, CARL M, KUMC,66103
588-7350
28 M 1003 56 R
MARTIN, NORMAN L, KUMC,66103
588-6800
36 M 1902 62 R
MARVIN, NORMAN G, KUMC 39TH AT RAINBDW,66103
588-6394
29 M 1902 56 FP
MASTERS, FRANCIS W, KU MED CENTER,66103
588-6142
20 M 3545 45 PS
MASTERSON, BYRON J, KUMC,66103
588-6216
33 M 2802 58 GYN
MATHEWSON, HUGH S, KUMC,66103
588-6675
21 M 1902 44 ANES
MATTIDLI, LEONE, KUMC,66103
588-6311
32 M 56115 56 PDC
MCCARTHY, ROBERT P, 155 S 18TH,66102
342-7233
25 M 2834 53 U
MC CUSKER, KEVIN T, KUMC, 66103
588-5000
51 M 1902 76 IM
MCGUIRE, MICHAEL R, 9119 WEST 74TH,66204
371-3515
43 M 1902 71 CD
MEBUST, WINSTON K, KU MED CENTER,66103
588-6148
33 M 5404 58 U
MEDEARIS SR, DONALD N, 3100 MINN AVE,66102
-
01 M 2401 27 DO
MEEK JR, JOSEPH C, KU MED CENTER,66103
588-5551
31 M 1902 57 IM
MESINA, ROLANDO R, 1102 MINNESOTA AVE,66102
381-3829
37 M 74801 61 GS
MILLER, HERBERT C, KUMC,66103
588-6300
07 M 801 34 PD

MINER JR, PHILIP B, KUMC 39TH AT RAINBDW,66103
588-6990
46 M 702 GE
MOELLER, DONALD D, 155 S 18TH,66102
371-4301
34 M 1902 60 IM
MOFFAT, ROBERT E, KUMC,66103
588-6800
42 M 1902 68 DR
MOORE, WAYNE V, KUMC,66103
588-6326
42 M 2604 70 PD
MORANTZ, ROBERT A, KUMC,66103
588-6119
42 M 3519 67 NS
MORFFI, RAUL R, 5411 LEAVENWORTH RD,66104
287-8300
25 M 27501 51 IM
MORRISON, RICHARD A, KU MED CENTER,66103
588-6815
34 M 1902 60 R
MULLEN JR, CLIFFORD J, 9201 PARALLEL,66112
334-4250
38 M 1902 64 OPH
MULLEN SR, CLIFFORD J, 1828 WASH BLVD,66102
281-3154
98 M 3006 23 OO
NASON, HERBERT M, 1225 NORTH 78TH,66112
299-1616
30 M 1902 56 FP
NEFF, JAMES R, KU MED CENTER,66103
588-6198
40 M 1902 66 ORS
NEIGHBOR, GAYLORD P, 1420 SOUTH 42ND,66106
588-1100
13 M 1902 41 FP
NEWKIRK, DARREL D, 619 ANN AVE,66101
321-4803
42 M 2101 67 PH
NOHE, PHILIP C, 266 NEW BROTHERHOOD BLDG,66101
321-4422
17 M 1902 42 ORS
NORRIS, CHARLEY W, KUMC,66103
588-6700
33 M 1902 64 OTO
NOTHNAGEL, ARNOLD F, 1610 WASHINGTON BOULEVARD,66102
342-1100
15 M 1902 39 GS
OLOFIELD, RAY W, 4631 ORVILLE,66102
334-6510
45 M 1902 72 FP
OTHMER, EKKEHARD, KUMC 39TH AT RAINBDW,66103
588-6440
33 M 40721 66 P
PARDO, LILLIAN G, KU MED CENTER,66103
588-6464
39 F 74807 62 PDN
PARDO, MANUEL P, KUMC 39TH AT RAINBOW, 66103
588-6464
35 M 74801 62 P
PAREKH, AJITKUMAR M, 860 NEWBROTHERHOOD BLDG,66101
-
47 M 49501 71 IM
PARK, CHAN H, KUMC 39TH AT RAINBOW, 66103
588-6029
36 M 58302 62 IM
PARRA, MIGUEL D, 6013 LEAVENWORTH RD,66104
299-2088
37 M 84710 64 FP
PASCHINO, ANTONIO R, 3500 RAINBDW,66103
432-3300
22 M 13201 60 P
PAZELL, JOHN A, 155 S 18TH ST,66102
371-6800
40 M 2501 66 ORS
PECANA, MANUEL C, BETHANY MED CENTER,66202
281-8896
45 M 74801 69 ANES
PERRY JR, LAWRENCE L, KUMC 39TH AT RAINBDW,66103
588-6522
34 M 1902 59 FP
PETELIN, JOSEPH B, 3227 N 54TH, 66104
588-6102
49 M 1902 76 GS
PETERS, GLENN R, 155 SOUTH 18TH,66102
371-6800
12 M 1902 37 GS
PIERCE, GEORGE E, KUMC 39TH AT RAINBOW,66103
-
33 M 2307 60 CD

PILCHARD, WILLIAM A, KU MED CENTER,66103
588-6600
39 M 1602 65 OPH
PISCHKE, FRANK J, 255 NEW BROTHERHOOD BLDG,66101
321-1161
36 M 1902 62 OTO
POL, P ALBERT, 175 W TERRACE TRAIL, 66106
588-6464
36 M 84710 67 P
PORTER, DAVID M, 4517 TROUP,66102
287-8800
39 M 4707 64 PD
POTTER, ROBERT L, 1969 N 33RD,66102
321-0341
38 M 1902 64 IM
POWERS, G ROBERT, 9201 PARALLEL,66112
299-0130
33 M 1902 65 FP
PREMSINGH, NALINI G, 860 NEW BROTHERHOOD BLDG, 66104
299-2088
39 F 49508 65 CO
PRESTON, DAVID F, KUMC,66103
588-6810
33 M 3841 59 NM
PRETZ, JAMES B, 1610 WASHINGTON BOULEVARD,66102
342-2442
24 M 1902 47 FP
PROUD, G ONEIL, KU MEDICAL CENTER,66103
588-6700
13 M 2802 39 OTO
PUGH, DAVID M, KUMC,66103
588-6015
29 M 801 58 CO
QUAISON, EMMELINE P, KUMC,66103
588-6463
45 F 74802 69 P
QUINN, CHARLES E, 1 GATEWAY CTR 4TH AND ST,66101
321-3355
43 M 4707 68 OBG
RECKLING, FREDERICK W, KUMC,66103
588-6129
34 M 3545 59 ORS
REDDY, EASHWER K, KUMC
588-7350
44 M 49597 68 TR
REDFORD, JOHN W B, KUMC,66103
588-6777
28 M 6501 53 PM
REGIER, HENRY L, 2000 WASHINGTON BOULEVARD,66102
81 M 1902 07 OO
RETTENMAIER, ALBERT J, 251 NORTH 15TH STREET,66102
96 M 3006 26 OO
RHODES, JAMES B, KU MEDICAL CENTER,66103
588-6019
28 M 1902 58 GE
RHODES, MARTIN L, 1969 N 33RD,66102
321-0341
47 M 1902 72 IM
RICE JR, FREDERICK A, 1029 N 32ND ST,66102
281-5252
36 M 4812 63 ORS
RICK JR, GREGORY GP, 155 S 18TH ST,66102
371-4301
40 M 1902 66 IM
RILEY, RAY B, 2020 ORVILLE,66102
06 M 1902 36 OO
RISING, JESSE D, KU MEDICAL CENTER, 66103
588-7130
14 M 1902 38 IM
ROBINSON, DAVID W, KU MEDICAL CENTER,66103
588-6136
14 M 4101 38 PS
ROBINSON, JOHN D, KUMC 39TH AT RAINBOW,66103
588-6670
48 M 1902 74 ANES
ROBINSON, RALPH G, KU MEDICAL CENTER,66103
588-6810
37 M 1902 62 NM
ROBISON, JAMES T, KUMC,66103
588-6600
21 M 4812 45 OPH
RODRIGUEZ, RAUL G, 3500 RAINBOW,66103
588-6463
42 M 26401 68 P
ROOK, LEE E, 4116 STRONG,66106
831-2834
09 M 1902 38 FP
ROSENTHAL, STANTON J, KUMC,66103
588-6800
46 M 1902 71 OR
ROTH, ALAN E, BETHANY HOSPITAL 51 N 12,66102
281-8814
35 M 1902 62 PATH
RUBIN JR, BEN, 132 SOUTH 19TH,66102
371-2561
37 M 3005 61 PD
RUTH, WILLIAM E, KU MED CENTER,66103
588-6044
26 M 1902 53 PUO
RYAN, MICHAEL J, 764 NEW BROTHERHOOD BLDG,66101
342-7070
14 M 2834 37 OTO
SAVIN, MICHAEL A, KU MED CENTER,66103
588-6935
44 M 4112 69 IM
SAVIN, VIRGINIA J, KU MED CENTER,66103
588-6969
44 F 4112 70 IM
SCHAUM, STEPHEN P, 4517 TROUP,66102
287-8800
44 M 1902 70 PD
SCHIMKE, R NEIL, KU MED CENTER,66103
588-6043
35 M 1902 62 IM
SCHLOERB, PAUL R, KU MED CENTER,66103
588-6110
19 M 3545 44 GS
SCHOTLAND, EDWARD S, 1300 N 78TH ST,66112
334-5250
36 M 86901 66 DERM
SCHREPFER, ROSEMARY, KU MED CENTER,66103
588-6290
22 F 1902 47 OBG
SCHWORM, CURTIS P, 155 SOUTH 18TH,66102
371-4343
47 M 3005 73 R
SERERES, EDGAR P, 662 NEW BROTHERHOOD BLDG,66101
321-2501
15 M 1902 39 FP
SHERMAN, ROBERT P, 9201 PARALLEL AVE,66112
334-0040
34 M 1902 63 IM
SIBALA, JUSTO L, KUMC,66103
588-6831
20 M 74802 49 R
SIFERS, EARL C, 155 S 18TH,66102
371-2900
24 M 1902 47 GS
SILVERGLAT, MICHAEL J, KUMC,66103
588-6464
43 M 1902 72 P
SILVERS, ALVIN, 1702 SW BOULEVARD,66103
236-9691
18 M 1902 45 FP
SMITH, STEPHEN D, KUMC 39TH AT RAINBOW,66103
588-6300
45 M 3006 71 PD
SNOW JR, ARTHUR D, KUMC,66103
588-7533
45 M 1902 75 FP
SOUCEK, CHARLES D, 155 S 18TH,66102
371-4343
31 M 3005 56 R
SPAULDING, JOHN S, KU MED CENTER,66103
588-5919
29 M 1602 59 PD
SPEER, LELAND, 1022 HOEL PARKWAY, 66102
12 M 1902 36 OO
STECHSCHULTE, DANIEL J, KUMC,66103
588-6008
36 M 2834 62 IM
STEELE, CLARENCE H, 255 BROTHERHOOD BLDG,66101
321-1161
14 M 1902 40 OTO
STEINZEIG, SHERMAN M, 155 S 18TH,66102
621-1151
25 M 1902 52 CD
STEPHENS, RONALD L, KUMC,66103
588-6029
39 M 1902 65 IM
STEVENSON, CHARLES E, KUMC,66103
588-6670
19 M 1902 44 ANES
STEWART, DAVID R, KUMC,66103
588-6124
38 M 3545 64 PD

STITT, RONALD W, 155 S 18TH,66102
342-7233
21 M 1902 45 U
STRICK, FRANK J, 7058 WAVERLY CT,66102
371-6841
17 M 1902 48 FP
STUBBLEFIELD, CHARLES T, 155 S 18TH,66102
371-2330
32 M 1902 58 OBG
TAYLOR, THOMAS L, 155 S 18TH ST,66102
371-2900
40 M 1902 66 GS
TEMPLETON, ARCH W, KUMC 39TH AT RAINBOW,66103
588-6805
32 M 3005 57 R
THEROU, LEONA F, KUMC,66103
588-5908
41 F 6701 67 PD
THOMAS, JAMES H, KUMC,66103
588-6115
41 M 2012 66 GS
THOMAS, THOMAS V, 4631 ORVILLE,66102
321-0145
37 M 49549 61 TS
THOMPSON, DANNIE M, ONE GATEWAY CENTER,66101
321-3355
35 M 4707 64 OBG
TOALSON, WILLIAM B, 155 S 18TH ST,66102
371-3515
37 M 1902 63 CO
TRUEWORTHY, ROBERT C, KU MED CENTER,66103
588-5701
40 M 2802 66 PD
TUCKER, SHERIDAN G, 3838 RAINBOW APT 1004,66103
-
50 M 1902 75 P
VALK, WILLIAM L, KU MED CENTER,66103
588-6146
09 M 2501 37 U
VAN THULLENAR, PHILIP A, BETHANY MED CENTER,66102
281-8834
31 M 2834 57 PATH
VARGHESE, GEORGE, KUMC 39TH AT RAINBOW,66103
-
44 M 49509 69 PM
VATS, TRIBHAWAN S, KUMC,66103
588-5701
40 M 49529 63 PD
VOPAT, RICHARD L, 4037 CAMBRIDGE, 66103
-
54 M MDST
WADDELL, BILL D, 155 SOUTH 18TH,66102
321-0386
31 M 3901 56 IM
WALKER, JACK O, KU MED CENTER,66103
588-6510
22 M 1902 53 FP
WALKER, MAURICE A, 3214 STRONG AVE,66103
831-1433
04 M 1601 28 OO
WATERS III, CHESTER H, KUMC,66103
588-6131
49 M 3005 75 ORS
WAXMAN, DAVID, KUMC,66103
588-5207
18 M 3515 50 IM
WEIGEL, JOHN W, KUMC,66103
588-6148
29 M 1902 54 U
WERTH, CLAUDE J, 4121 W 83RD,66208
334-0040
37 M 1902 64 P
WEST, C OMER, 414 HURON BLDG,66101
371-3572
92 M 1902 23 OO
WILLIAMS JR, STERLING B, KUMC,66103
588-5000
41 M 401 73 OBG
WILLIAMSON, JOHN A, 3900 RAINBOW KUMC,66103
588-5000
51 M 1902 76 ORS
WILSON, SLOAN J, 5618 W 62ND,
-
10 M 1902 36 OO
WINTER JR, CALVERT J, 132 S 17TH,66102
342-5372
22 M 1902 47 PD
WOLF, KARL T, 621 NORTHRUP AVE,66101
-
14 M 1902 48 OO

WOLKOFF, A STARK, KUMC,66103
588-6219
21 M 4109 52 OBG
WONG, BERT Y, KUMC,66103
588-6015
40 M 801 65 IM
WRIGHT JR, ROBERT W, 669 NEW BROTHERHOOD BLDG,66101
371-5344
24 M 1902 48 GS
YULICH, JOHN O, 2106 CHESTER COURT,66106
384-1630
33 M 1902 59 FP
ZIEGLER, DEWEY K, KU MED CENTER,66103
588-6985
20 M 2401 45 N
ZINN, THOMAS W, 155 S 18TH,66102
371-4343
41 M 1902 67 R
ZORNOZA, JESUS, KUMC 39TH AND RAINBOW,66103
588-6886
47 M 84704 70 R

KINGMAN—316
(Pratt-Kingman Society)

BLOOM, L THEIL, 708 N MAIN,67068
532-3147
32 M 1902 57 R
BOYER, ROBERT E, 349 N MAIN,67068
532-3171
36 M 1902 63 FP

KINSLEY—316
(Edwards County Society)

ATWOOD, M DALE, 616 NILES,67547
659-2114
19 M 1902 51 FP
MCKIM, W LYNN, 109 WEST 8TH,67547
659-2137
33 M 1902 59 FP
SCHNOEBELEN, RENE E, 807 EAST 4TH,67547
659-2141
16 M 3901 40 FP

KIOWA—316
(Tri-County Society)

CHRISTENSEN, MARION D, 802 DRUMM,67070
825-4121
25 M 3901 52 FP

LA CROSSE—913
(Central Kansas Society)

SERRANO, JOAQUIN, MEDICAL CLINIC PO BOX 459,67548
222-2564
29 M 27501 56 FP

LANSING—913
(Leavenworth County Society)

SNOW, DONALD L, HOLIDAY PLAZA,66043
727-3204
21 M 64901 54 OBG

LARNED—316
(Pawnee County Society)

BRENNER, WILLIAM R, 804 CARROLL,67550
285-3133
15 M 3006 39 FP
CRAM JR, OLE R, 722 MANN,67550
285-2141
18 M 1902 43 FP
DAVIS, DAVID M, 815 W 6TH,67550
285-2131
04 M 1902 30 P
EWING, THOMAS O, 804 CARROLL,67550
285-3133
22 M 1902 46 FP
PATTERSON, ALICE M, PO BOX 7,67550
-
13 F 1902 38 OO
PATTERSON, HAROLD L, PO BOX 7,67550
825-3636
13 M 1902 38 OO
*SHAH, MIAN, 313 W 14TH,67550
-
M FP
*SHAH, NASREEN, 313 W 14TH,67550
-
F OBG
SHEPARD, LEROY W, 603 W 5TH,67550
-
03 M 3006 30 OO

SMITH, JOHN D, 804 CARROLL,67550
285-3133
22 M 3901 51 FP

LAWRENCE—913
(Douglas County Society)

ANDERSON, WINSTAN L, 3603 W 10TH ST,66044
—
09 M 1902 34 OO
AUCHARD, VIRGIL M, 2126 LOUISIANA,66044
—
89 M 1902 23 OO
BAILEY, WILLIAM A, 404 MAINE ST,66044
843-9125
40 M 1902 66 ORS
BELL, MARGARET E, PROF BLDG,66044
841-3535
37 F 1902 63 CHP
BELOT JR, MONTI L, LAWRENCE NATIONAL BK BLDG,66044
843-6340
13 M 1902 40 FP
BIERI, PETER V, 944 KENTUCKY,66044
841-5217
45 M 1902 71 ENT
BISHOP, RODNEY L, 346 MAIN, 66044
—
49 M 1902 75 IM
BITTENBENDER, LEE R, 930 IOWA,66044
842-7001
46 M 1902 72 O
BLAIR, T RICHARD, 404 MAINE ST,66044
842-3635
34 M 1902 60 IM
BOYDEN, MARY S, MEDICAL ARTS BLDG,66044
842-3778
14 F 2604 38 PDA
BRANSON, VERNON L, 346 MAINE,66044
842-4477
17 M 1902 42 PD
BRAY, E DEAN, 346 MAINE,66044
842-5070
29 M 1902 60 FP
BRUNER, STEVEN C, 500 ROCKLEDGE,66044
841-6540
46 M 1902 72 FP
BUCK JR, HENRY W, MEDICAL ARTS BLDG,66044
843-0677
34 M 1902 60 OBG
BUTTON, JESSE H, 935 IOWA ST,66044
841-3535
35 M 1902 63 P
CHEDIAK, ELIAS, 601 MISSOURI,66044
841-7430
39 M 84704 65 P
CLARK, MARGARET G, HILLCREST MED CENTER,66044
843-3888
10 F 1902 44 FP
CLARK, RAY A, 2103 KINGSTON,66044
—
07 M 1902 39 OO
CLINTON, DALE L, CITY HEALTH DEPT,
841-5716
21 M 1902 54 PH
CULVER, WARREN T, 2500 W 6TH,66044
842-4178
20 M 3508 46 DPH
DUNLAP, RICHARD L, MEDICAL ARTS CENTER,66044
842-4344
12 M 3005 37 ENT
FRIESEN, DALE, 2500 WEST 6TH,66044
842-7026
47 M 1902 74 ANES
GILLES, HELEN M, MED ARTS CENTER,66044
842-4477
22 F 1902 45 PD
GODWIN, PHILLIP A, 500 ROCKLEDGE,66044
842-1070
28 M 1902 55 ANES
GRAY, CAPTAIN K, MED ARTS BLDG, 66044
842-7200
48 M 1902 75 IM
HATTON, DONALD W, 404 MAINE ST,66044
842-3635
42 M 1902 68 IM
HERMES, RICHARD L, THE MEDICAL ARTS CENTER,66044
843-0677
15 M 4112 39 OBG
HIEBERT, DAVID L, 2209 HILL COURT,66044
841-3211
36 M 1902 61 R
HIRD, WAYNE E, 329 MISSOURI,66044
842-4300
26 M 1902 50 TS
HUGHES, ROBERT WALTER, 346 MAINE,66044
843-1374
27 M 1902 54 FP
INGHAM JR, H LAIRD, 404 MAINE,66044
842-3635
45 M 3901 70 IM
JONES, H PENFIELD, MED ARTS CENTER,66044
842-0211
06 M 2401 31 GS
JOSEPH, HOWARD F, DOCTORS BUILDING,66044
843-3981
26 M 1902 51 U
KIMBROUGH, VICTORIA F, 935 IOWA,66044
843-8425
10 F 4706 37 DPH
LEARNED, GEORGE R, 401 ARKANSAS,66044
843-5502
22 M 1902 55 GS
LESSENDEN, GLENN A, MED ARTS BLDG,66044
843-6233
24 M 1902 48 FP
MADSEN, GLENN L, 404 MAINE,66044
841-3211
38 M 3005 65 R
MANAHAN, G EUGENE, MED ARTS CENTER,66044
842-0211
19 M 1902 44 GS
MOORELL, CARL A, 404 MAINE,66044
843-3680
45 F 1902 71 PATH
NELSON, RICHARD O, 201 W 8TH,66044
843-0921
11 M 1001 41 FP
OELSCHLAGER, RONALD D, 404 MAINE,66044
841-3211
43 M 1902 69 R
OLSON, CARL E, 107 WEST 7TH,66044
842-9911
17 M 1611 46 FP
ORCHARD, RICHARD A, 932 MASSACHUSETTS ST,66044
841-2280
41 M 2802 68 DPH
OWEN, GARRY D, 4TH & MAINE,66044
843-0677
37 M 1902 63 OBG
*PRAEGER, MARK R, 944 KENTUCKY,66044
843-2010
42 M 1902 68 GS
PRICE JR, LAURANCE W, 404 MAINE ST,66044
843-3680
33 M 1902 59 PATH
REED, RALPH R, 404 MAINE,66044
842-3635
27 M 1902 53 IM
REESE, JOHN L, 4TH & MAINE,66044
842-6644
35 M 1902 61 GS
ROBERTS, RICHARD S, 308 MAINE,66044
843-6137
19 M 2802 44 GS
ROBISON, CORBIN E, 901 KENTUCKY ST,66044
843-3033
14 M 1902 39 GS
SANDERS, J ALAN, LAWRENCE MEMORIAL HOSP,66044
842-2083
29 M 1902 60 PATH
SCHROEDER, SYDNEY D, WATKINS MEMORIAL HOSP,66044
864-4045
18 M 1902 44 P
SCHWEGLER, RAYMOND A, 1504 UNIVERSITY DR,66044
843-4455
07 M 2604 31 OBG
*THOMSEN, STEVEN T, 500 ROCKLEDGE,66044
844-6540
47 M 3005 77 FP
TOTTEN, FREDERICK E, WATKINS MEMORIAL HOSP, 66045
843-4455
17 M 1902 50 IM
WALTERS, BYRON W, 1030 AVALON RD,66044
843-6751
13 M 1902 39 FP
WELL, MICHAEL A, 944 KENTUCKY,66044
843-3981
41 M 1606 67 U

WERTZBERGER, JOHN, 404 MAINE ST.66044
843-9125
36 M 1902 63 ORS
WILCOX SR, HOWARD L, MEDICAL ARTS CENTER.66044
843-0677
18 M 3520 44 OBG
WOLLMANN, MARTIN, 2615 ORCHARD LN.66044
843-9530
26 M 1902 57 IM
WRIGHT, M ERIK, 2728 STRATFORD ROAD.66044
-
1S M 502 50 P

LEAVENWORTH—913
(Leavenworth County Society)

ANWAR, M ZIA, 109 DELAWARE.66048
682-6661
39 M 11801 67 IM
BARRY, DAVID R, 500 EISENHOWER RD.66048
727-6000
42 M 1902 68 FP
BENNETT, CHARLES A, MEDICAL ARTS BUILDING.66048
96 M 1902 25 OO
CENAC, MARK T, 500 EISENHOWER RD.66048
727-6000
27 M 401 49 GS
CHANGPONG, SALA, 500 EISENHOWER RD.66048
727-6000
46 M 89103 70 OBG
COMBS, G RALPH, CUSHING HOSP PAVILLION.66048
-
79 M 4101 02 OO
COMBS, PETER S, 213 DELAWARE.66048
682-0127
14 M 4101 41 IM
DUYSAK, SAMI, 520 6TH AVE.66048
682-6661
22 M 90201 47 IM
GERBER, HARRY A, 605 N 6TH.66048
682-3893
96 M 5606 30 OO
GERJARUSAK, PRAPAS, 500 EISENHOWER RD.66048
727-6000
46 M 89101 71 IM
GRAHAM, J MALCOLM, 500 EISENHOWER RD.66048
727-6000
28 M 3840 54 PD
GRAHAM, KENNETH L, 500 EISENHOWER RD.66048
727-6000
21 M 3840 45 GS
GRAHAM, THOMAS W, 500 EISENHOWER RD.66048
727-6000
26 M 3840 50 IM
GRISOLIA, ANDRES, 424 WALNUT ST.66048
682-5400
27 M 84708 50 OS
HAMMEKE, JOHN C, 3601 S 4TH ST TRAFFICWAY.66048
682-5201
27 M 401 61 OP
JOHNSON, PAUL O, MEDICAL ARTS BLDG.66048
682-6661
36 M 1902 61 FP
MCCOLLUM, MARY B, 607 SHAWNEE.66048
651-6566
43 F 4804 68 P
MCCOLLUM, WILLIAM B, MEDICAL ARTS BLDG.66048
682-6661
41 M 1902 66 TS
MCKEE, RICHARD S, 422 WALNUT.66048
682-1023
05 M 1902 35 AN
MERRITT, W HENRY, 520 6TH AVE.66048
682-6661
14 M 702 39 GS
PALMER, MARVIN M, 500 EISENHOWER RD.66048
727-6000
45 M 702 71 OBG
PARKER, ROBERT W, 500 EISENHOWER RD.66048
727-6000
45 M 1902 71 FP
PRATT, WILLIAM L, 901 6TH AVE.66048
-
05 M 3006 30 OO
RABE, MELVIN A, 520 SIXTH AVE.66048
682-6661
14 M 1902 37 GS
STRUTZ, WILLIAM C, 68 WESTWOOD DR.66048
682-8868
08 M 5606 43 R
VOORHEES, CARROLL O, 520 SIXTH AVE.66048
682-6661
25 M 1902 52 FP
VOORHEES, GORDON S, MEDICAL ARTS BUILDING.66048
642-6661
12 M 1902 39 IM

LEAWOOD (see Shawnee Mission)

LEBO KS 316
-
HUNTER, KENNETH R, .66856
2S6-256S
07 M 1902 39 FP

LENEXA (see Shawnee Mission)

LENORA—913
(Northwest Kansas Society)

STEICHEN, EDWARD F, .67645
05 M 1601 31 OO

LIBERAL—316
(Seward County Society)

ALLEN, RAY E, 2 PLAZA DR.67901
624-5691
37 M 1902 64 IM
CADDO, CARMELITA O, PO BOX 1643 SW MED CENTER.67901
624-1651
41 F 74801 63 R
CAMPION, WOODROW M, 121 W THIRD.67901
624-2594
13 M 1902 39 IM
GRIMES, I ROSS, 222 W 15TH.67901
624-1676
27 M 3901 54 TS
HARRIS, NORVAN O, 222 W 15TH.67901
624-3811
20 M 1902 44 OBG
HOLCOMB, DONALD G, 15 E 11TH.67901
-
12 M 1902 38 GS
HOLCOMB, WILLIAM M, 15 E 11TH.67901
624-2252
31 M 3901 56 GS
KOONS, JESS W, 1210 N WASHINGTON.67901
624-3841
27 M 1902 57 OPH
NEVINS, RICHARD L, 610 WEST 11TH.67901
624-1841
47 M 3901 73 FP
PALMER JR, H C, 2 PLAZA DRIVE.67901
624-5691
36 M 1902 63 IM
PROCHAZKA, OTTO F, PO BOX 1067.67901
624-1651
12 M 1902 38 R
RATHBUN, EDWIN O, 610 W 11TH.67901
624-1841
36 M 1902 62 FP
REESE, JACK O, 15 E 11TH.67901
624-6226
32 M 1902 57 FP
SEAGO, CHARLOTTE L, 1114 N WASHINGTON.67901
624-1625
35 F 1720 60 PD
WADE, THEODORE E, 318 NORTH LINCOLN.67901
04 M 512 30 OO

LINCOLN—913
(Central Kansas Society)

MADDEN, WILLIAM J, 115 W SPRING.67455
524-5154
24 M 1902 51 FP
SONGER, HERBERT L, .67455
524-4053
12 M 1902 38 FP

LINDSBORG—913
(McPherson County Society)

FREDRICKSON, DUANE E, 121 W LINCOLN,67456
227-3371
39 M 1902 66 FP
FULLER, DERYL D, 121 W LINCOLN, 67456
227-3371
25 M 1902 50 FP
MURFITT, MALCOLM C, 231 N MAIN,67456
227-2732
13 M 801 41 FP

LOUISBURG—913
(Miami County Society)

KULLMAN, FREDERICK M, PO BOX 760,66053
837-2911
45 M 702 71 FP

LYNDON—913
(Franklin County Society)

STOUT, NILES M, ,66451
828-4521
16 M 1902 50 FP

LYONS—316
(Rice County Society)

GARWOOD, JAN D, 1221 W NOBEL, 67554
257-5124
49 M 1902 74 FP
GRIMES, JAMES T, 1221 W NOBLE,67554
257-5124
27 M 1902 53 FP
SIEMENS, RICHARD A, 1221 W NOBLE,67554
257-5124
30 M 1902 59 FP
WOLF, CURTIS V, 1221 W NOBLE,67554
257-5124
37 M 1902 64 FP

MANHATTAN—913
(Riley County Society)

BAKER, RICHARD B, 1133 COLLEGE,66502
537-4200
42 M 4113 68 ORS
BALL, RALPH G, 215 S DELAWARE,66502
—
03 M 1902 27 OO
BASCOM, GEORGE S, 1133 COLLEGE,66502
539-6574
27 M 2401 52 GS
BOESE, KENNETH M, 1133 COLLEGE AVE,66502
776-4744
25 M 1902 56 FP
BROWN, ROBERT M, LAFENE ST HEALTH CENTER,66502
532-6544
31 M 1902 63 FP
BUROICK, BRUCE M, MANHATTAN PSYCH CLINIC,66502
776-9411
25 M 512 53 P
CATHEY, ROBERT H, 215 SOUTH WIND,66502
537-4990
42 M 1902 68 O
CRANE, C HERBERT, 1133 COLLEGE,66502
537-9030
22 M 3520 46 PO
DOUTHIT, JOHN O, 2950 NEVADA,66502
—
37 M 3005 62 ORS
DURKEE, WILLIAM R, 1133 COLLEGE AVE,66502
776-4744
23 M 1902 45 IM
FAIRCHILD, JOHN A, 1133 COLLEGE,66502
539-4041
14 M 3006 41 ANES
FISCHER, REX R, 1133 COLLEGE,66502
539-5322
34 M 3005 60 OBG
FREEMAN, FRED A, MANHATTAN MED CENTER,66502
537-8710
42 M 1902 69 U
GARDNER, JAMES D, 1133 COLLEGE AVE,66502
537-4940
43 M 2834 71 IM
HEASTY, ROBERT G, 1133 COLLEGE,66502
539-5322
11 M 3519 38 OBG
HOSTETTER, PHILIP H, 821 POYNTZ,66502
537-2544
17 M 1902 42 FP
HUNTER JR, JAMES S, 1133 COLLEGE,66502
539-5501
18 M 2301 41 OBG
JUBELT, HILBERT P, 1133 COLLEGE,66502
537-9030
19 M 1611 43 PO
KALOOR, RICHARD H, PO BOX 128,66502
539-5363
40 M 2401 66 PATH
KEMPTHORNE, CHARLES R, 519 N 11TH,66502
—
03 M 5605 32 OO
KIRK, THOMAS E, 1133 COLLEGE AVE,66502
537-0655
44 M 3005 71 OPH
KLINGLER JR, EUGENE A, 1133 COLLEGE,66502
539-5341
35 M 1902 62 GS
LAFENE, BENJAMIN W, 1844 ANDERSON AVE,66502
—
01 M 3806 31 OO
LOWE, STANLEY W, 1133 COLLEGE AVE,66502
776-3451
32 M 1902 59 OPH
LYONS JR, FRANK C, 1133 COLLEGE AVE,66502
539-7641
44 M 3840 70 OR
MARSHALL, RONALD L, 1133 COLLEGE AVE,66502
539-5322
42 M 3005 67 OBG
MARTIN, DANIEL C, LAFENE HEALTH CENTER KSU,66506
532-6544
30 M 1902 58 IM
MCKNIGHT, DAVID E, 1133 COLLEGE AVE,66502
539-7641
32 M 1902 62 R
MCNEIL, ELBERT O, 1133 COLLEGE,66502
537-9030
22 M 702 48 PD
MEEK, PALMER F, 1133 COLLEGE,66502
537-2651
45 M 1902 71 IM
MILLER, ABRAHAM H, 1133 COLLEGE,66502
527-2651
29 M 4101 54 IM
MOSIER, STEVEN J, 1133 COLLEGE AVE,66502
776-4744
49 M 1902 74 FP
MOWRY, GERALD L, 1133 COLLEGE,66502
539-5322
26 M 1902 53 OBG
OLNEY, ROBERT O, 1133 COLLEGE AVE,66502
539-7555
27 M 3005 51 GS
PETERSON, JACK T, 1133 COLLEGE,66502
539-5363
25 M 1902 50 PATH
PHILLIPS, STEPHEN B, LAFENE ST HEALTH CENTER,66506
532-6544
17 M 1902 45 IM
REITZ, LELAND C, 1133 COLLEGE,66502
539-3501
36 M 1902 63 IM
REITZ, ROGER P, 1133 COLLEGE,66502
539-3501
32 M 1902 59 IM
ROSE, GRAHAM C, 215 S WIND,66502
537-9030
46 M 4706 70 PD
RYAN, THOMAS F, 404 HUMBOLDT ST C,66502
—
M FP
SINCLAIR, ROBERT E, LAFENE ST HEALTH CENTER,66502
532-6544
24 M 3840 52 AOM
STEIN, ROBERT B, 1133 COLLEGE,66502
539-1851
29 3515 52 P
STONE, G REX, 1133 COLLEGE AVE,66502
539-7555
29 M 1902 54 GS
TIEMANN, WILLIAM H, 1133 COLLEGE,66502
532-6544
42 M 3005 67 FP
VOLKMANN II, HARLEY W, 1133 COLLEGE AVE,66502
539-7641
47 M 1902 72 R

WHITE, THADDEUS H. 1735 ANDERSON AVE.66502
-
1S M 1902 42 00

MANKATO—913
(Republic County Society)

KIMBALL, RICHARD R. 102 S CENTER.66956
378-3511
45 M 1001 72 FP
SCHLOTTERBACK, WILLIAM E. PO BOX 193.66956
378-3511
31 M 1902 61 FP

MARION—316
(Marion County Society)

ENSEY, T CRANFORD. 504 S ROOSEVELT.66861
382-2182
20 M 4804 47 FP
GUERRA, TOMAS H. 122 S CEDAR.66861
382-2137
33 M 84704 65 IM

MARYSVILLE—913
(Northeast Kansas Society)

ARGO, DONALD A. 810 N 19TH.66508
562-2303
36 M 3005 64 FP
LAWS, LEWIS R. 810 N 19TH.66508
443-3121
25 M 1902 54 FP

McLOUTH—913
(Jefferson County Society)

SNOOK, ROBERT RUFUS. .66054
796-6116
11 M 1902 42 FP

McPHERSON—316
(McPherson County Society)

BILLINGS, THOMAS. 400 WEST 4TH.67460
241-5500
39 M 1902 66 FP
BRANDSTED, ERNEST C. 400 W 4TH.67460
241-1654
18 M 1606 44 OBG
COLLIER, WILLIAM J. 400 W 4TH.67460
241-1766
25 M 3605 49 TS
DYCK, ARTHUR H. 101 1/2 NORTH MAIN.67460
03 M 1902 28 00
JOHNSON, J RICHARD. 400 W 4TH.67460
241-4293
28 M 1902 55 IM
PIERSON, W WEIR. 823 N MAIN.67460
241-1445
17 M 1902 44 FP
PRICE, VAUGHAN C. 120 N ASH.67460
05 M 4706 29 00
SOHLBERG JR, ROBERT. 116 N ASH.67460
241-2321
05 M 1606 34 FP

MEADE—316
(Iroquois County Society)

DAUGHERTY, ROBERT M. 631 E WALNUT.67864
-
09 M 1902 36 00
DONAHOE, DORVAL H. PO BOX 488.67864
873-2181
32 M 05501 63 FP
HILL, RICHARD H. 234 EAST CARTHAGE.67864
873-2113
18 M 1902 44 FP

MEDICINE LODGE—316
(Tri-County Society)

HOFFER, JOHN G. 604 N WALNUT.67104
886-3222
13 M 1902 44 GS
STUCKY, DEAN E. 116 E KANSAS.67104
886-5653
33 M 1902 60 FP

MERRIAM (see Shawnee Mission)

MINNEAPOLIS—913
(Saline County Society)

WEDEL, KENNETH D. 311 N MILL ST.67467
392-2144
32 M 1902 60 FP
WEDEL, KERMIT G. 311 N MILL ST.67467
392-2144
32 M 1902 60 FP

MINNEOLA—316
(Iroquois County Society)

STEPHENS, CHARLES. MINNEOLA CLINIC.67865
885-4202
33 M 2803 58 FP
*TROTTER, CORKY R G. BOX 567.67865
-

MISSION (see Shawnee Mission)

MORAN—316
(Allen County Society)

NEVITT, J RUSSELL. P O BOX 430.66755
-
99 M 3510 32 00

MOUNDRIDGE—316
(McPherson County Society)

KAUFMAN, WILLARD E. 115 N CHRISTIAN AVE.67107
345-6322
28 M 1902 53 FP
LOGANBILL, VARDEN J. 115 N CHRISTIAN AVE.67107
345-6322
26 M 1902 54 FP

MULVANE—316
(Sedgwick County Society)

COBB, LESLIE H. 102 E MAIN.67110
777-1441
17 M 4804 47 FP

NASHVILLE—316
(Pratt-Kingman Society)

WAYLAN, THORNTON L. .67112
06 M 1902 35 00

NEODESHA—316
(Southeast Kansas Society)

CHRONISTER, BERT. PO BOX 118.66757
325-2622
38 M 1902 64 FP
MOORHEAD JR, F ALLEN. 709 MAIN ST.66757
325-2200
39 M 1902 65 FP

NESS CITY—913
(Central Kansas Society)

WIENS, PETER K. BOX 8.67560
798-3191
24 M 1902 55 FP

NEWTON—316
(Harvey County Society)

ALLEN, FRANCES A. 1112 BOYD.67114
1S F 1902 43 00
BENTON, JAY S. WOMENS MEDICAL CENTER PA.67114
283-3600
23 M 4804 49 OBG

CAMPBELL, FRANCES S, 1901 E FIRST,67114
283-2400
35 F 4101 61 P
CARPER, IVAN H, AXTELL CLINIC,67114
283-2800
28 M 1902 59 GS
CARPER, OWEN E, AXTELL CLINIC,67114
283-2800
37 M 1902 64 FP
CLAASSEN, MILTON A, 201 S PINE ST,67114
283-3600
32 M 1902 58 ORS
CLARK, LOELLA E, AXTELL CLINIC,67114
283-2800
42 F 512 73 FP
CRAIG, CHARLES C, 3 HAWTHORNE CT,67114
-
45 M 1902 71 ORS
CRANSTON, STEPHEN O, 203 EAST BROADWAY,67114
-
71 M 1902 71 GS
ENNS, EUGENE K, 6 INDIAN LANE,67114
-
15 M 1902 40 OO
ENNS, JAMES H, 900 N POPLAR,67114
283-1400
24 M 1902 47 OPH
FENT, LEE S, 316 OAK,67114
283-0505
14 M 2834 43 GS
FRANSEN, HERBERT, 209 S PINE,67114
283-5040
32 M 6501 60 GS
FRANSEN, PAUL H, 209 S PINE,67114
283-5040
46 M 6501 71 FP
GLOVER, RICHARD M, 203 E BROADWAY,67114
283-2800
21 M 1902 53 FP
GRISWOLD, DALE G, 203 E BROADWAY,67114
283-2800
27 M 1902 53 IM
GROVE, JOHN A, AXTELL CLINIC,67114
283-2800
08 M 1606 37 ORS
HAWKEY, ALFRED S, 203 E BROADWAY,67114
-
03 M 1902 29 OO
HILL, PHILLIP K, BETHEL CLINIC,67114
283-3600
29 M 1803 56 IM
HWA, EUGENE C, BETHEL CLINIC,67114
283-1160
21 M 24216 47 R
ISAAC, CHARLES A, AXTELL CLINIC,67114
283-2800
25 M 1902 49 U
KLASSEN, DANIEL S, 316 OAK,67114
283-1313
13 M 1902 42 OBG
KLIEWER, VERNON L, MENTAL HEALTH CENTER,67114
283-2400
31 M 1606 57 CP
MYERS, ROBERT W, RT 1,67114
-
11 M 1902 43 OO
NACHTIGALL, ANDREW, BETHEL CLINIC,67114
283-3600
28 M 1902 59 PD
OLSON, ERWIN T, BETHEL CLINIC,67114
283-3600
19 M 1902 47 PD
OTOOLE, JAMES K, PRAIRIE VIEW HOSPITAL,67114
283-2400
28 M 1643 54 P
PEDRAZA, HERNANDO V, 4 SYCAMORE CT,67114
264-1381
28 M 26404 56 R
PREHEIM, DELBERT V, 209 S PINE,67114
283-5040
13 M 702 42 IM
QAMAR, YUSUF, AXTELL CLINIC,67114
283-2800
38 M 70409 62 IM
RADOVANOV, RADMILA, 500 MAIN,67114
-
34 F 95702 60 R
RICH, ELDON S, BETHEL CLINIC,67114
283-6000
16 M 1902 46 GYN
ROULEAU, DAVID N, 209 S PINE,67114
283-5040
47 M 1902 73 FP
SCHMIDT, HERBERT R, CEDAR VILLAGE,67114
-
03 M 1902 34 OO
SILLS, CHARLES T, AXTELL CLINIC,67114
283-2800
09 M 1902 37 IM
TANDOC JR, VALENTIN T, BETHEL CLINIC,67114
283-3600
39 M 74809 62 U
TOMPKINS, CARL O, 316 OAK STREET,67114
283-1380
22 M 1902 51 FP
VAUGHAN, DONNA A, 201 S PINE,67114
283-3600
45 F 1902 71 IM
VOGT, VERNON W, BETHEL CLINIC,67114
283-3600
22 M 3005 53 FP
VONGKASEMSIRI, SUNAN, 500 NORTH MAIN,67114
283-1160
41 M 89101 67 R
WEBER, ROY R, 209 S PINE,67114
283-5040
46 M 1902 73 IM
WIENS, J WENDELL, 201 S PINE,67114
283-3600
32 M 1902 59 GS

NORTON—913
(Northwest Kansas Society)

COLIP, F MERLYNN, 711 N NORTON,67654
927-3305
35 M 1902 61 FP
COOPER, ARTHUR E, 305 W WILBERFORCE,67654
-
08 M 1611 34 OO
HARTLEY, ROY W, 711 N NORTON,67654
927-3305
37 M 1902 63 FP
HARTMAN, ROGER L, 711 N NORTON,67654
927-3305
35 M 1902 61 FP
LONG, ROBERT C, 711 N NORTON,67654
927-3305
27 M 1902 53 GS

NORTONVILLE—913
(Jefferson County Society)

MADISON, WILLARD A, ,66060
886-2110
20 M 1902 51 FP

OAKLEY—913
(Northwest Kansas Society)

OHMART, RICHARD V, PO BOX 756,67748
672-3262
36 M 1902 62 FP
SEKAVEC, GORDON B, 209 CENTER AVE,67748
672-3351
07 M 1902 38 FP

OBERLIN—913
(Northwest Kansas Society)

WHITAKER, REN R, OBERLIN CLINIC,67749
475-2222
37 M 5404 66 FP

OLATHE—913
(Johnson County Society)

ARONOFF, MICHAEL E, 105 PATRONS PLAZA BLDG,66061
782-3953
39 M 1604 64 ENT
BARE, CHARLES E, 540 E SANTA FE,66061
782-2020
43 M 1902 69 U
BEEBE, EDMER, 420 EAST CEDAR,66061
-
03 M 5605 32 OO
BLISS, JOY V, OLATHE COMMUNITY HOSP,66061
782-2292
42 F 3005 68 AN

BROWN, PAUL W. 405 CLAIRBORNE,66061
782-3322
44 M 1902 70 FP
CHENOWETH, JOHN R, O.D., 1003 STRATFORD RD,66061
782-2442
36 M 2878 70 OST
DELPHIA, ROBERT E. 401 CLAIRBORNE,66061
782-1610
24 M 1902 56 FP
EIDT, DAVID W. 407 CLAIRBORNE,66061
782-8487
44 M 2501 70 FP
EIOT, LAURENCE A. 407 CLAIRBORNE,66061
782-8487
44 M 1902 71 FP
FORTUNE, CEDRIC B. 405 CLAIRBORNE,66061
782-3322
40 M 1902 66 FP
GLAZZARO, CHARLES D. 407 CLAIRBORNE,66061
782-3384
28 M 2507 56 P
HALVORSON, HOWARD C. 407 CLAIRBORNE,66061
782-2020
41 M 5404 66 U
HUDSON, ROBERT P. 12925 FRONTIER RD,66061
588-7040
26 M 1902 52 IM
JENSEN, THOMAS M. 407 CLAIRBORNE,66061
782-1148
47 M 3005 73 ORS
LAIRD, DALE O. ONE PATRONS PLAZA,66061
782-3631
42 M 1902 68 OPH
MATTHEW, WILLIAM L. 405 CLAIRBORNE,66061
782-3322
29 M 1902 56 FP
MCCANN, WILLIAM E. OLATHE CLINIC,66061
782-0262
22 M 3901 48 FP
MEE, ADRIAN W. 300 S ROGERS RD,66061
782-2292
19 M 1902 54 ANES
MILLIGAN, DONALD B. 401 CLAIRBORNE,66061
782-1610
48 M 2307 74 FP
MORGAN, DAVID LLOYD II, 807 CLAIRBORNE,66061
782-8300
49 2820 77 IM
PIERRON, GEORGE J. 540 EAST SANTA FE,66061
782-0260
22 M 1902 47 FP
PINCOMB, ARTHUR L. 401 CLAIRBORNE,66061
782-1415
20 M 1902 51 FP
RATNAM, DASARI V. 539 EAST SANTA FE,66061
782-2100
38 M 49500 60 P
ROMONDO, STEVEN A. 300 SOUTH ROGERS RD,66061
782-2292
47 M 1902 73 ANES
RUHLEN, JAMES L. 807 CLAIRBORNE,66061
782-8300
46 M 1902 72 IM
SEAMAN, LAUREN I. 401 CLAIRBORNE,66061
782-1415
07 M 6001 38 FP
SETTLE, JR, RUSSELL O, 407 CLAIBORNE, 66061
—
35 M 1902 60 P
SHEFFER, KEITH O. 407 CLAIRBORNE SUITE 5,66061
782-1148
37 M 1720 67 ORS
SOELONER, JAMES O. 405 CLAIRBORNE SUITE 2,66061
782-8600
44 M 1902 70 FP
STOPPLE, JOHN A. 405 CLAIRBORNE SUITE 4,66061
782-3073
42 M 5605 69 OBG
TRIMBLE, DAVID P JR, 540 EAST SANTA FE,66061
764-3081
37 M 401 65 IM
YEOMANS, RONALD N, 405 CLAIRBORNE SUITE 4,66061
782-3073
40 M 1902 67 OBG

ONAGA—913
(Pottawatomie County Society)

FLECKENSTEIN, CHARLES S. PO BOX 125,66521
07 M 1902 36 00

WALSH, EUGENE A. ,66521
889-4241
18 M 3006 42 FP
WALSH, THOMAS E. ONAGA CLINIC,66521
889-4241
48 M 1902 74 FP

OSAGE CITY—913
(Flint Hills Society)

WILLIAMS, HOMER J. 524 MARKET STREET,66523
528-3211
05 M 1902 31 FP

OSAWATOMIE—913
(Miami County Society)

APPENFELLER, WILLIAM O. 524 BROWN AVE,66064
755-3166
25 M 1902 53 FP
MORALES, AMALIA O. P O BOX 500,66064
755-3151
22 F 2150I 50 ADM
MORALES, OTTO E. P O BOX 500,66064
755-3151
22 M 2750I 51
QUINONES, ELADIO A. OSAWATOMIE STATE HOSP,66064
755-3151
16 M 2750I 43 FP

OSBORNE—913
(Central Kansas Society)

KOZNEK OD, CLARE, P O BOX 177,66066
863-2142
47 M 2878 76 GP
LEE O O, ROBERT PAUL, 520 ATCHISON,66066
863-2142
44 M 2878 GP

OSWEGO—316
(Labette County Society)

BURGESS, ARTHUR P, 504 5TH STREET,67356
795-4427
19 M 1902 52 FP

OTTAWA—913
(Franklin County Society)

GOLLIER, ROBERT A, MED ARTS BLDG,66067
—
13 M 1902 37 DO
GOLLIER II, ROBERT A, MEDICAL ARTS BLDG,66067
242-1620
40 M 1902 66 FP
HADLEY, DELMONT C, 1320 SOUTH ASH,66067
242-3891
35 M 1902 64 FP
HENNING, CALVIN W, 1502 CEDAR,66067
242-2641
05 M 1902 35 00
LAURY, DAVID G, 1320 S ASH,66067
242-1620
17 M 1606 44 FP
LOFGREEN, VICTOR J, ROUTE #1,66067

97 M 1902 32 00
PHILGREEN, DONALD E, 1320 S ASH,66067
242-3891
39 M 1602 67 FP
REYES JR, FRANCISCO A, 1320 S ASH,66067
242-5312
38 M 74801 61 GS
SAYLOR, STEPHEN, 1320 ASH,66067
242-1620
47 M 1902 73 FP
SPEER, LOUIS N, PO BOX 0,66067
242-1257
14 M 1606 41 FP
STREHLOW, CHESTER H, MEDICAL ARTS BUILDING,66067
242-3891
30 M 1902 57 FP
SUTTON, ROBERT E, 1320 S ASH,66067
242-1620
46 M 1902 72 FP

OVERBROOK—913
(Shawnee County Society)

RUBLE JR, JAMES L, OVERBRDDK CDMM CL1N1C,66524
665-2205
26 M 1902 S3 FP

OVERLAND PARK (see Shawnee Mission)

PAOLA—913
(Miami County Society)

BANKS, ROBERT E, PO BDX 298,66071
294-230S
29 M 1902 SS FP
NAIK, GCPAL V, 700 8APTIST E DR,66071
-
M 49S01 DBG
RDWLETT, JACK G, PO DRAWER A,66071
294-23S6
21 M 1902 52 FP
STANLEY, REX C, PO DRAWER A,66071
294-23S6
24 M 1902 52 GS

PARSONS—316
(Labette County Society)

AVES, AGNES M, 1S09 MAIN ST,67357
421-0600
38 F 74801 59 1M
AVES, RENATO B, 1509 MAIN STREET,673S7
421-0600
3S M 74801 59 GS
BAIR, HOWARD V, 2601 GABRIEL,67357
421-65S0
18 M 1902 43 P
BDRKLUND, MAURICE K, 400 KATY,67357
421-2700
21 M 1720 50 GS
CRAMER, GUY W, 412 MURDOCK,673S7
-
11 M 1902 39 OD
DEBRIERE, SIDNEY L, 2601 GABRIEL,67357
421-6550
19 M 702 52 PD
HENDERSDN, CHARLES F, 1509 MAIN,67357
421-0600
14 M 1902 40 A
LAVAVESHKUL, CHIRUND, 107 MAIN PO BOX 290,67357
421-6210
40 M 89102 63 GS
LIUJULUSCHAN, RIT, 107 MAIN,67357
421-6210
41 M 89101 68 GS
MARTIN, EARL A, 1S16 GRAND,673S7
-
07 M 1606 35 OO
MILLER, CHARLES H, 2819 CLARK,67357

07 M 3006 32 OO
MILLER, DEAN M, 108 CRESTVIEW,67357
421-4280
22 M 1902 48 R
PACE, JDHN D, 11S 1/2 S 18TH ST,67357
421-1010
94 M 1902 20 OD
PARANJOTHI, SUBRAMONIAM P, 1509 MAIN,67357
421-6160
39 M 49S31 64 1M
PAULS, DANIEL N, 1710 CORNING,67357
421-1431
45 M 1902 71 1M
ROTHSTEIN, TERRY B, 220 NORTH 32ND,673S7
421-S900
43 M 1606 69 DPH
SWARTZ, WARREN E, 400 KATY AVE, 67357
421-2700
2S M 1902 52 GS
TANGCHUPONG, CHANTRASIRI, 310 N 16TH,67357
421-2460
47 F 89102 71 PD
TANGCHUPONG, SAROHD, 310 N 16TH,67357
421-2460
43 M 89102 69 OBG
WHITE, JOHN P, PARSDNS CL1N1C,673S7
421-0600
17 M 1902 42 FP

PHILLIPSBURG—913
(Central Kansas Society)

RICHARDS, JON F, PHILLIPS CO MED CLINIC,67661
S43-5211
50 M 1902 7S FP

PITTSBURG—316
(Crawford County Society)

ARMSTRONG, HARDLD J, PROFESSIONAL BUILDING,66762
232-2600
40 M 1902 68 ORS
BENA, JAMES H, 109 EAST 9TH,66762
231-69S0
12 M 300S 36 PD
BERKEY, VERNDN A, KIRKWOOD BLDG,66762
231-7650
18 M 1902 43 R
BIERLEIN, KENNETH J, 812 S CATALPA, 66762

06 M 1606 33 OD
BRAUN, WILLIAM T, 214 ELMWOOD DR,66762
-
10 M 4706 35 OO
CARSDN, RICHARD CARLYLE, MT CARMEL MEDICAL CENTER,66762
-
3S M 1720 63 A
COOMER, TYLER E, 315 NATL BANK BLDG,66762
231-7730
30 M 2101 59 GS
COOPER, KENT J, 2308 TUCKER TERR,66762
231-6280
41 M 1902 73 FP
ERICKSON, CLARENCE W, 217 NATL BANK BUILDING,66762
231-7400
06 M 1902 33 IM
ESCH, JCHN G, 207 KIRKWOOD BLDG,66762
231-5360
24 M 3006 48 GS
ESPEDIDO-RAMIRZ, IRENE P, 2308 TUCKER TERR,66762
231-6280
43 F 74801 67 PD
GOMEZ, MODESTO S, 2308 TUCKER TERRACE,66762
231-2490
3S M 72601 63 PD
GREGORY, ARLENE, MT CARMEL MED CENTER,66762
231-6100
36 F 1102 66 ANES
HOLSINGER, DONALD M, 1015 MT CARMEL PL,66762
231-5900
38 M 1902 64 1M
HUERTER, DAVID F, 2308 TUCKER TERR,66762
231-16S0
46 M 1902 72 1M
LANCE, RAYMDND W, 608 W QUINCY, 66762
22 M 1902 47 OD
LEFFLER, PAUL B, 309 WINWOOD,66762

02 M 1902 40 CO
LYONS, DAVE J, 107 W 4TH,66762
231-5160
07 M 2834 30 FP
MCKEE, DICK B, 1S00 S COLLEGE,66762
-
96 M 1902 28 OO
MILLER, EARL E, 1312 S BRDADWAY,66762
231-6410
13 M 1902 37 EENT
MULLER, SAMUEL B, 611 W QUINCY,66762
-
05 M 1902 34 DO
NEIGHBDR, ERNEST H, SE KS DRTHOP CLINIC,66762
232-1600
40 M 1902 66 DRS
NEWMAN, CLIFFORD B, 1204 E 7TH,66762
-
01 M 1902 28 OO
ODGERS, RDDNEY K, 909 CENTENNIAL, 66762
49 M 1902 74 IM
PARSI, MANUTCHEHR K, 2308 TUCKER TERR,66762
231-3770
38 M S1701 64 OBG
POGSDN, GEORGE W, 1015 MT CARMEL PLACE,66762
231-5900
24 M 1902 47 1M
RAMIREZ, AUGUSTD H, 2308 TUCKER TERR,66762
231-1600
32 M 26407 58 1M

SCHLEMMER, ROGER B, 1009 S BROADWAY,66762
231-6380
37 M 1902 68 OPH
TWEET, FREDERICK A, MT CARMEL HOSPITAL,66762
231-6100
39 M 1602 66 PATH
WOOD, DOUGLAS H, 413 W JEFFERSON,66762
—
11 M 5605 36 OO
YAGHMOUR, TALOOT E, 905 N LOCUST, 66762
231-0850
40 M 33002 64 U
ZABEL, KENNETH P, 2308 TUCKER TERR,66762
231-1650
37 M 1902 65 IM

PLAINVILLE—913
(Central Kansas Society)

PAGE, O VALE, 409 S COCHRAN ST,67663
434-4609
20 M 1902 51 FP
PEDERSON, ARNOLD M, PLAINVILLE CLINIC,67663
434-4609
22 M 1902 51 FP

PLEASANTON—913
(Bourbon County Society)

JUSTUS, WILLIAM J, ,66075
352-6134
29 M 1902 55 FP

PRAIRIE VILLAGE (see Shawnee Mission)

PRATT—316
(Pratt-Kingman Society)

AMBLER, CARL D, 200 COMMODORE,67124
—
M 1902 57 R
BARKER, PATRICK N, 1219 E FIRST,67124
672-5666
45 M 1902 71 GS
BLACK, CYRIL V, 223 E 4TH,67124
672-6403
05 M 4802 30 GS
FILLEY, VERNON W, 310 E 2ND,67124
672-5555
13 M 3005 43 GS
FREEMAN, F GILES, 310 E 2ND,67124
672-5555
18 M 1902 44 FP
JACKS, J WARREN, 602 E 2ND,67124
672-5559
23 M 1902 48 FP
PITMAN, WILL D, 717 WEST 3RD,67124
—
98 M 1902 25 OO
QUENZER, RONALD W, 1219 EAST 1ST,67124
672-5666
46 M 1601 73 IM
THOMAS, R CULLEN, 1219 E FIRST,67124
672-5666
47 M 1902 72 GS
THORPE, FRANCIS A, 310 E 2ND,67124
672-5555
08 M 1606 35 FP
WOLFF, FREDERICK P, 223 E 4TH,67124
672-6403
20 M 1902 44 IM

PROTECTION—316
(Iroquois County Society)

GLENN, LYLE G, 146 BRDADWAY BOX 447,67127
622-4686
12 M 1606 40 FP

QUINTER—913
(Northwest Kansas Society)

CARMAN, DEAN R, QUINTER CLINIC,67752
754-3333
36 M 1902 69 FP
GUNTER, CARL C, QUINTER CLINIC BLDG,67752
754-3333
20 M 1902 51 FP
HIESTERMAN, HERMAN W, QUINTER CLINIC BLDG,67752
754-3333
23 M 1902 51 FP

RANSOM—913
(Central Kansas Society)

MCLAIN, KENNETH, BOX 247,67572
731-2295
21 M 1902 46 FP

RILEY—913
(Riley County Society)

OLTMAN, THEODORE V, PO BOX 68,66531
—
00 M 1601 29 OO

RUSSELL—913
(Central Kansas Society)

MERKEL, EARL O, SHIELDS BLDG,67665
483-2178
32 M 1902 57 FP
PANICHABHONGSE, SAMBHUNDH, 213 WEST 7TH,67665
483-2178
43 M 89101 67 GS
PETTIJOHN, WALTER J, 231 E 15TH,67665
—
12 M 1902 37 OO
STARKEY, JERALD L, SHIELDS BUILDING,67665
483-2178
30 M 1902 56 FP
SWANN, CLAIR L, 112 W 7TH,67665
483-4212
13 M 1902 39 IM
WHITE, FAGAN N, 356 W 5TH,67665
—
11 M 702 36 OO

SABETHA—913
(Northeast Kansas Society)

BROWN, VIRGIL E, 1018 MAIN ST,66534

06 M 1902 37 OO
JUMPER, JERRY L, D.O., 824 MAIN ST,66534
284-3483
42 M 2878 70 OST
MONTGOMERY, THOMAS ALLEN, 1018 MAIN ST,66534
284-2141
10 M 1902 49 FP

SALINA—913
(Saline County Society)

ALLEN, MONTE L, 600 S SANTA FE,67401
827-0307
36 M 1902 61 OTO
ANDERSON, JOOY, 737 E CRAWFORD,67401
827-7261
32 F 1902 59 IM
BAXTER, W REESE, 600 S SANTA FE,67401
825-8221
47 M 1902 73 FP
BRUMMETT, RICHARD R, P O BOX 176,67401
325-2622
34 M 1902 64 FP
BRUNGARDT, BERNARD A, 400 E BELOIT,67401
827-4433
21 M 3006 46 ANES
CALDERWOOD, WILLIAM A, PO BOX 918,67401
827-9631
41 M 1902 68 FP
COFFEY, ROY B, 671 ELMORE DRIVE,67401
823-6397
24 M 1902 47 ORS
COVERT, THOMAS J, 737 E CRAWFORD,67401
827-7261
45 M 1902 71 PD
CULTRON, FRANK T, 800 E CRAWFORD,67401
823-8151
10 M 1643 38 OPH

OOWELL, JAMES C, 645 E IRON,67401
827-7255
26 M 1611 49 IM
ORAEMEL, H RICHARD, 600 S SANTA FE ST,67401
827-0307
18 M 1902 53 OTO
OREMER, HENRY S, 737 E CRAWFORD,67401
827-7261
18 M 1902 43 IM
EATON, GLEN E, RFO #3,67401
827-3064
28 M 1902 54 ANES
EATON, LESLIE F, 737 E CRAWFORD,67401
827-7261
06 M 1902 32 GS
ELLISON, PAUL O, 1499 E IRON,67401
825-7271
35 M 2105 60 OPH
FEIGHNY, ROBERT E, 519 S SANTA FE,67401
827-4424
20 M 1902 51 ORS
FORSTER JR, LOUIS G, 617 E ELM,67401
825-8221
47 M 1902 73 FP
FREEMAN, RAYMOND S, 737 E CRAWFORD,67401
827-7261
20 M 702 50 PO
GANS, FREDERICK A, 737 E CRAWFORD,67401
227-7261
22 M 2834 46 PEO
GOERING, OONALD O, 617 E ELM,67401
825-8221
31 M 1902 56 FP
GRIFFING, RICHARD B, 1941C GLENDALE RD,67401
825-9091
27 M 1902 53 ANES
GUNN, MARVIN R, 617D UNITED BLDG,67401
827-9526
28 M 3901 54 R
HARRIS, NORMAN R, 430 S 7TH,67401
825-8191
30 M 1902 59 OBG
HARTENBOWER, CAREY A, 1515 E IRON,67401
827-9526
20 M 1902 51 OO
HATTON, LLOYD W, 406 UNITED BLDG,67401
827-6256
06 M 1902 33 P
HODGES, MERLE A, 430 S 7TH ST,67401
825-8191
34 M 1902 58 OBG
HOLMAN, JON B, 326 NORTH FRONT,67401
827-9366
33 M 1902 63 P
JACKSON JR, DELMAS A, 645 E IRON,67401
827-7255
35 M 2101 60 IM
JENKINS, NEAL M, 135 E CLAFLIN,67401
827-9631
19 M 1902 49 IM
LAKE, MAX S, 600 S SANTA FE,67401
827-0307
19 M 3005 43 OPH
LASLEY, DAVID A, 645 E IRON, 67401
827-9635
22 M 1606 47 U
LUNGSTRUM, JACK E, 645 E IRON,67401
823-2215
21 M 1902 59 ORS
MACY, NORMAN E, 400 SOUTH SANTA FE,67401
827-4053
35 M 1902 60 PATH
MACY, TED L, PO BOX 360,67401
827-7261
43 M 1902 71 GS
MARCHBANKS, OONALD L, 1911-E GLENDALE RD,67401
823-2380
24 M 1902 51 FP
MARSHALL, GEORGE W, 430 S 7TH,67401
225-3191
44 M 1902 70 OBG
MARTIN, OLIVER L, 135 E CLAFLIN,67401
827-9631
08 M 1902 37 OBG
MATHIS, JERRY L, 1112 ALBERT,67401
827-9343
35 M 1902 62 POA
MAXWELL, GORDON E, 135 E CLAFLIN,67401
827-9631
29 M 1902 55 OBG

MCCRAE, SPENCER C, 519 S SANTA FE,67401
827-4424
18 M 3509 43 ORS
MILLER, ELDEN V, 1928 RIDGELEA,67401
827-3061
19 M 1902 44 ANES
MITCHELL, JOHN C, 617 UNITED BLDG,67401
827-3081
13 M 1902 38 GS
MOWERY, WILLIAM E, 737 E CRAWFORD,67401
823-6455
23 M 1902 47 GS
NICKELL, WENDELL K, 617 UNITED BLDG,67401
827-9618
26 M 1606 50 TS
NULL, WILLIAM G, 135 E CLAFLIN,67401
827-9631
31 M 102 57 PO
OBANDO, GUILLERMO, 2110 KNOLLCREST DR,67401
823-6219
35 M 26404 62 R
PALMER, GERALD K, C/O ST JOHNS HOSP BX 1285,67401
-
24 M 1803 53 PATH
RASSA, REZA P, 204 SEITZ DRIVE,67401
827-4411
33 M 51701 59 R
REECE, RICHARD J, 617 D UNITED BLDG,67401
827-9526
23 M 1902 49 R
RODERICK, JAMES E, 645 E IRON,67401
827-9635
23 M 1902 47 U
ROMEISER, REX S, 645 E IRON,67401
827-9635
41 M 1902 67 U
RUEB, ANDREW E, 11 CRESTVIEW DR,67401
827-6691
11 M 1606 35 GS
SCOTT, CHESTER E, 519 S SANTA FE,67401
827-5549
23 M 1902 51 FP
SEBREE, STEVEN G, PO BOX 360,67401
827-7261
47 M 1902 73 OBG
SIMPSON, J COLBERT, 308 WEST SOUTH,67401
-
07 M 3006 38 OO
SLOO, MILO G, PO BOX 1346,67401
823-2215
41 M 1902 67 ORS
SMITH, HAROLD R, 737 E CRAWFORD,67401
827-7261
19 M 1902 51 GS
TAYLOR, THOMAS F, 430 S OHIO,67401
827-0346
26 M 1902 53 FP
WAGENBLAST, HOWARD R, 737 E CRAWFORD,67401
827-7261
21 M 1902 49 FP
WATERS, CLARENCE N, 530 S 5TH,67401
823-6497
13 M 2834 48 O
WEBER, CLARENCE J, 400 S SANTA FE,67401
-
00 M 1902 39 OO
WEBER, ROBERT W, 645 E IRON,67401
827-7255
26 M 1902 49 IM

SCOTT CITY—316
(Southwest Kansas Society)

ARROYO, ZEFERINO, SCOTT CITY CLINIC,67871
872-2187
M 74802 GS
FIELDS, GALEN W, 202 COLLEGE,67871
872-2187
15 M 1902 49 FP
HOPKINS JR, B MORRIS, PO BOX 97,67871
872-2187
23 M 1902 53 FP

SEDAN—316
(Southeast Kansas Society)

TAYLOR, ELMER W, 120 WEST OSAGE,67361
725-3141
28 M 512 57 FP

WALKER, WILLIAM K, 111 E CHEROKEE,67361
725-3171
18 M 1902 45 FP

SENECA—913
(Northeast Kansas Society)

BERKLEY, NORMAN W, 1S SOUTH 5TH ST,66538
336-2128
31 M 1902 63 FP
GILBERT, J HOWARD, S22 MAIN,66538
336-2414
05 M 1902 41 00

SHARON SPRINGS—913
(Northwest Kansas Society)

CHUNG, JOHN J, WALLACE CO MED CLINIC,67758
852-4214
23 M 58301 48 FP

SHAWNEE MISSION (Mission, Leawood, Lenexa, Prairie Village, Overland Park)—913
(Johnson County Society)

ALLEN, MAX S, KUMC 39TH AT RAINBOW,66103
588-6988
11 M 1902 37 IM
ALLEN, TIMOTHY E, 6742 FONTANA,66208
—
49 M 1902 RES
ALLEN JR, LEWIS G, 7301 MISSION RD SUITE 300,66208
362-2222
21 M 1902 45 R
ALTENBERND, ELVIN CONRAD, 7319 W 81ST,66204
648-2010
26 M 1902 54 FP
ALVAREZ, LUIS A, 8100 MARTY,66204
649-3400
32 M 64914 61 FP
APPELBAUM, ANN H, 8645 OVERHILL RD, 66206
—
24 F 3506 50 P
ARMBRUSTER, ALBERT A, 9119 WEST 74TH SUITE 202,66204
362-9220
17 M 512 55 GS
ATHON, MERRILL O, 8501 DELMAR,66207
642-4242
24 M 1902 54 FP
BAEKE, JOHN D, 6806 WEST 83RD,66204
642-4242
19 M 1902 52 FP
BALANDFF, ARNOLD Z, 4601 W 109TH SUITE 122,66211
642-4040
42 M 1803 67 PD
BANSAL, SATISH C, 6100 MARTWAY,66202
384-2220
38 M 49541 61 ORS
BARKER, ELIZABETH B, 4121 WEST 83RD SUITE 123,66208
381-6669
30 F 4706 55 P
BARKER, JAMES BERTON, 8686 W 96TH SUITE 200,66212
381-3338
31 M 4706 55 DTO
BARNHART, RONALD J, 9119 WEST 74TH,66204
831-2334
41 M 2501 68 OBG
BARR, RICHARD N, 7301 MISSION ROAD,66208
432-4366
32 M 1902 57 OPH
BARRICK, BRUCE, SH MISSION MEDICAL CENTER,66201
676-2340
39 M 1902 65 PATH
BARRY, JOHN WILLIAM, 5510 WEST 84TH,66207
—
22 M 1902 46 R
BATTY, THOMAS V, 5555 W 58TH,66202
432-2080
21 M 3806 54 FP
BELL, DELORIS J, 4601 WEST 109TH SUITE 104,66211
341-6550
42 F 1902 68 OPH
BELZER, EDWARD G, 4601 WEST 109TH SUITE 110, 66211
381-8282
36 M 3005 58 PD

BILLINGSLEY, THAD H, 6335 ASH,66202
649-5566
41 M 1902 66 P
BILLINGSLEY JR, JOHN A, 10302 WEST 62ND APT E7,66203
585-1177
31 M 1902 58 FP
BISHOP, FRANCIS E, 7501 MISSION ROAD, 66208
648-3533
20 M 1902 45 P
BLETZ, DONALD B, 6100 MARTWAY,66202
362-7290
28 M 5104 58 IM
BOLES, J MICHAEL, 5949 NIEMAN ROAD,66203
631-1300
35 M 1902 61 FP
BOSILJEVAC, FRED N, 5301 MISSION WOODS ROAD,66205
342-4843
16 M 1902 44 OPH
BRIDGENS, JAMES G, SH MISSION MEDICAL CENTER,66201
676-2340
22 M 1902 47 PATH
BROWN, WILLIAM R, 7301 MISSION RD,66208
236-8866
23 M 1902 48 IM
BUBB, STEPHEN K, 4934 HOWE DR,66205
588-5000
48 M 1902 74 ORS
BUCHNER, DAVID M, 6815 GRANADA LN,66208
—
51 M 1902 RES
BURGER, PAUL B, 5638 NIEMAN ROAD,66203
631-6114
25 M 2834 50 FP
CAIN, IVAN W, 4140 W 71ST ST,66208
362-7811
18 M 1902 44 ORS
CALDERON, JAIME, 7301 MISSION RD,66208
362-8566
39 M 26401 66 IM
CALKINS, LARRY L, JOHNSON CO NATL BK BLDG,66207
649-0110
18 M 1902 43 OPH
CAROUFF, JAY J, 6300 GLENWOOD,66202
432-4480
25 M 3006 54 FP
CAREY, LARRY J, 2624 WEST 77TH,66208
—
51 M 1902 RES
CARRASCO, LENORE C, 5811 OUTLOOK,66202
432-0625
41 F 74801 63 A
CAVITT, ROBERT F, 9119 WEST 74TH,66204
831-0700
24 M 1902 48 GS
CHANG, SHU FANG, 10200 W 75TH,66204
432-7885
25 F 24239 49 P
COE, RICHARD O, 7301 MISSION RD,66208
362-8505
31 M 4804 56 OPH
COHEN, ROBERT A, 3700 W 83RD,66208
642-2100
39 M 2803 64 PD
COHN, STEVEN G, 7907 WEST 99TH ST,66212
588-6670
41 M 1902 67 ANES
COOPER, JACK R, 7301 MISSION RD,66208
432-6300
17 M 3840 43 NS
CORBIN, MURRAY O, 6100 MARTWAY,66202
362-7290
39 M 1902 65 IM
CORDELL, LARRY O, 7301 MISSION RD,66208
362-8317
41 M 1902 67 ORS
COULTER, HENRY F, 5829 WOODSON PO BOX 975,66201
722-1100
23 M 1902 51 FP
COX JR, IRA, 5829 WOODSON PO BOX 975,66201
722-1100
19 M 1902 49 FP
CURRAN, KEVIN E, 4121 W 83RD,66208
649-9383
39 M 2803 65 OPH
DARR, RICHARD B, 74TH & GRANDVIEW,66204
676-2214
42 M 3401 70 IM
DAVIS, RICHARD E, 10955 GRANADA LN,66207
561-2025
26 M 1902 54 P

DELP, MAHLON H, 6131 TERRYDALE RD,66203
588-6976
03 M 1902 34 IM
DENISON, TERRY R, 5829 WCODSON ROAD,66202
722-1106
29 M 1902 56 PD
DERRINGTON, KENNETH L, FOX HILL MED BLDG,66211
341-3535
44 M 1902 71 FP
DIEHL, ANTONIO M, 13106 W 75TH TERR,66216
* -
24 M 2604 47 PDC
DOCKHORN, ROBERT J, 5300 W 94TH TERR, 66207
381-4674
34 M 1902 60 PDA
DOHERTY, WILLIAM R, 7600 STATE LINE,66208
649-3900
20 M 3006 56 FP
DONNELLY, FRANCIS M, 5830 HOWE DRIVE,66205
334-2500
48 M 1902 74 ANES
DUCKETT II, THOMAS G, 4601 W 109TH,66211
648-1022
41 M 1902 67 OPH
EIKERMANN, WILLIAM C, 9400 MISSION RD,66206
642-5184
42 M 1902 69 P
ENDERS, WRAY, 9034 COTTONWOOD DR,66215
-
02 M 1902 36 OO
ESRIG, HAROLD L, D.O., 8132 SAGAMORE,66206
381-5033
30 M 2878 60 ANES
ETZENHOUSER III, RUSSELL D, P O BOX 7426,66207
381-8282
34 M 1902 59 PD
EVANS JR, WILLIAM E, 7301 MISSION ROAD,66208
362-7363
24 M 1902 58 FP
FIXLEY, MARK S, KUMC 39TH AT RAINBOW,66103
-
45 M 1902 72 IM
FULLEN, JERYL G, 10000 W 75TH,66204
831-3500
43 M 401 68 ORS
GARCIA, FRANCISCO, 9409 W 82ND, 66204
276-4000
32 M 27501 60 FP
GARDNER, GLENN M, 5200 WEST 64TH,66208
-
35 M 2803 60 IM
GENTRY, KALE C, 6806 W 83RD,66204
632-4242
31 M 1902 60 FP
GILBERT, ROBERTA M, 4121 W 83RD,66208
341-1234
35 F 3506 62 P
GILLEN, BILLY A, 8802 BIRCH LANE,66207
381-0521
29 M 1902 54 ANES
GOLLERKERI, MOHAN P, 7301 MISSION RD, SUITE 339, 66208
236-8866
30 M 49516 52 HEM
GOMEZ, FRANCISCO, 4CCO SOMERSET DRIVE,66208
649-7300
15 M 26401 43 P
GOOD, WENDELL LISLE, 4601 W 109TH,66211
649-3883
24 M 1902 48 FP
GRADY, KENNETH L, 4121 W 83RD SUITE 150,66208
649-5566
36 M 1902 69 P
HAMTIL, LAWRENCE W, 3700 WEST 83RD,66208
649-6722
36 M 2803 61 PD
HARD, BENJAMIN F, 7301 MISSION RD,66208
362-2340
28 M 4802 55 OBG
HARMS, ALBERT C, 5750 WEST 95TH,66207
381-5550
13 M 1902 38 FP
HARNED, BERT W, SHAWNEE MISSION MED CNTR,66201
676-2479
24 M 1606 47 ANES
HARPSTER, GENE D, 4601 W 109TH,66211
648-1400
31 M 1902 57 GS
HASTINGS, MARY T, 2520 W 50TH PL,66205
-
45 F 1902 77 RES

HERZON, CHARLES D, 8951 W 75TH,66204
649-9010
39 M 3006 65 OBG
HESSER, HERBERT H, 7207 EDGEWOOD BLVD,66203
-
06 M 1902 34 OO
HIGGINS, RONALD A, 6100 MARTWAY,66202
362-7290
37 M 2803 65 D
HOBSON, MILBURN W, 9119 W 74TH ST,66204
831-2334
30 M 1902 55 OBG
HODES, HERBERT C, 4601 W 109TH SUITE 330,66211
381-6868
43 M 1902 69 OBG
HODGES, BRUCE E, 12345 W 95TH,66215
888-0777
32 M 1902 63 FP
HOPKINS, WILLIAM O, 10000 W 75TH,66204
831-3500
33 M 2803 61 ORS
HOPKINS JR, LENLY T, 8600 W 95TH ST,66212
649-7844
30 M 3841 56 GS
HORSEMAN, ROBERT F, 9119 W 74TH ST,66204
432-7419
19 M 1902 44 OBG
HYLWA, THEODORE M, 7301 MISSION RD,66208
362-7505
43 M 1902 69 PS
ITURRALDE, GEORGE, 7501 MISSION RD,66208
648-4949
21 M 13201 49 P
JANES, DONALD R, 4601 WEST 109TH,66211
381-6144
34 M 1902 60 OBG
JAYARAM, MARANDAPALLI R, 6100 MARTWAY,66202
299-8000
42 M 49509 65 PD
JOHLER, TERRY H, 6100 MARTWAY,66202
362-7290
40 M 2802 67 FP
JONES, CHARLES E, 74TH & GRANDVIEW,66201
676-2214
31 M 1902 60 FP
JONES, HIVOR, 8901 W 74TH SUITE 269,66204
381-1331
24 M 80303 51 P
JORG, HELEN H, 74TH & GRANDVIEW,66201
676-2479
21 F 2802 53 ANES
JOUVENAT, NEIL C, 11055 CEDAR,66211
341-6030
43 M 3005 71 OBG
KAGAN, STUART M, 7301 MISSION RD,66208
362-6000
44 M 4901 69 PD
KENNEDY, KENNETH R, 12827 W 78TH,66216
432-0126
24 M 1902 53 FP
KHURANA, SATISH K, 9119 W 74TH,66204
432-3334
41 M 49536 65 PD
KIMURA, CHARLES C, 5811 OUTLOOK,66202
432-0625
25 M 2101 56 A
KODANAZ, A AYTEKIN, 5710 REINHARDT DR,66205
334-2500
28 M 90201 55 ANES
KOZIKOWSKI, BEN M, 7301 MISSION RD,66208
362-8317
30 M 2834 55 ORS
KUBIN, DORIS A, 2504 W 71ST,66208
371-4343
15 F 1902 43 R
KURTH, ROBERT H, 5555 W 58TH,66202
432-2080
28 M 3005 53 IM
LAMBRIGHT, ROBERT L, 2108 WEST 75TH,66208
262-6380
26 M 4705 50 P
LAPI, RUTH M, 2012 STRATFORD RD,66208
-
14 F 4107 37 OO
LEATHERS III, HOLLIS K, SHAWNEE MISSION MED CNTR,6620
676-2340
38 M 3901 64 PATH

LEGASPI JR, PEDRO L, SHAWNEE MISSION MED CNTR,66201
831-8592
36 M 74801 60 ANES
LEIGH, LAWRENCE E, 8020 SANTA FE DR,66204
642-4080
12 M 1902 41 FP
LEWIN, WALTER, 8901 W 74TH ST S 269,66204
362-4040
30 M 1902 56 P
LEWIS, JAMES E, 3700 W 83RD SUITE 203,66208
649-0923
37 M 2101 63 P
LIPSEY, JAMES H, 10000 W 75TH SUITE 103,66204
831-3500
31 M 1606 56 ORS
LOEWEN, PETER S, SALEM HOSPITAL, 66205
-
91 M 1902 30 DD
LOTITO, CARLOS A, 7301 MISSION RD,66208
362-8566
29 M 13201 56 IM
LULO, ANTONIO R, 7600 STATE LN #150,66208
649-3900
35 M 30801 60 IM
MALLORY, JOHN A, 6100 MARTWAY,66202
362-7290
43 M 2803 71 IM
MANLEY, JOSEPH W, 4601 W 109TH SUITE 306,66211
381-8838
42 M 1902 69 OBG
MANTZ, FRANK A, 9309 W 103RD,66212
588-7070
12 M 4101 38 PATH
MASER, GEORGE R, 5808 NALL,66202
432-5515
12 M 1902 36 FP
MATHEWS, ROBERT MAJOR, 7301 MISSION RD,66208
362-6888
25 M 1902 54 GS
MAXWELL, ROBERT A, 9119 W 74TH,66204
432-9119
46 M 1902 73 PO
MCCAUGHEY, HUGH W, 11055 CEDAR SUITE 210,66211
381-1724
28 M 1902 53 IM
MCEACHEN, WILLIAM H, 3700 WEST 83RD SUITE 102,66208
649-3335
32 M 1902 59 PD
MC GUIRE, MICHAEL RAY, 9119 W 74TH, 66204
371-3515
43 M 1902 71 CO
MCGURK, THOMAS E, 4601 W 109 SUITE 125,66211
649-2080
39 M 2803 65 P
MCWHERTER, LOTTIE B, 5920 NALL SUITE 308,66202
362-1464
30 F 1902 57 IE
MELENDRES, JUANITO M, 8971 W 75TH,66204
341-2000
36 M 74809 61 PD
MELIA JR, B JAMES, 3700 W 83RD,66208
648-4117
33 M 2803 60 O
MENENDEZ, JOSE D, 8100 MARTY,66204
649-3400
25 M 27501 54 P
MENEZ, CESAR V, 4121 W 83RD SUITE 120,66208
381-4484
36 M 74810 56 P
MERCER, JAMES B, 13109 WEST 95TH,66215
888-5442
21 M 1902 51 FP
MORCOS, ABDELMALAK E, SHAWNEE MISSION MED CNTR,66201
831-8592
33 M 33004 61 ANES
MUEHLBERGER, JAMES J, 4601 W 109TH SUITE 314,66211
383-3222
34 M 3006 60 PD
MUELLER, J KENT, 3700 WEST 83RD SUITE 203,66208
649-0923
35 M 1902 62 P
NASH, ROBERT A, 4601 W 109TH,66211
649-8686
31 M 1902 55 P
NEIBURGER, JAMES B, 5300 W 94TH TERR,66207
381-4674
46 M 1642 72 A
NEIGHBOR, ERNEST G, 3617 JOHNSON DRIVE,66106
588-1100
06 M 1902 33 FP

NIEMAN, JOHN L, PO BOX 7426,66207
381-8282
28 M 3806 58 PD
NOSTI, JUAN C, 7301 MISSION RD, SUITE 345, 66208
262-5014
38 M 13204 63 PS
NUNEZ, JULIAN, 10021 MASTIN,66212
642-5000
30 M 27501 60 FP
NYE, C ERIK, 7301 MISSION RD SUITE 348,66208
-
39 M ORS
O'BRYAN, JAMES J, 3700 W 83RD, 66208
649-9500
47 M 1902 73 PD
O'CONNELL, FRANK A, 7830 STATE LINE,66208
381-0886
25 M 1902 51 FP
O'DELL, MICHAEL L, 9256 COUSER APT 1A,66212
341-6376
51 M 1902 MDST
OKTAWIEC, DANUTA, 5848 FONTANA DRIVE,66205
236-6261
22 F 80303 50 ANES
OSGOOD, GEORGE M, 3700 WEST 83RD,66208
381-5200
14 M 1902 44 IM
PATTERSON, JOHN R, 5317 CHADWICK RD,66205
-
20 M 1902 48 PD
PEARCE, EUGENE W J, 9119 WEST 74TH STREET,66204
831-2334
24 M 2802 49 OBG
PEARCE, LUNETTA M, 9119 W 74TH,66204
362-1525
26 F 3005 49 FP
PENTECOST, RICHARD L, 8900 STATE LINE SUITE 350,66206
383-1410
32 M 1001 56 P
PETERSEN, A GENE, 3700 W 83RD,66208
648-3911
27 M 1902 54 IM
PETERSEN, GERALD D, 3700 WEST 83RD,66208
648-3911
30 M 1902 60 IM
PETRIE, SAMUEL C, 6100 MARTWAY, SUITE 228, 66202
722-1166
27 M 1902 58 IM
PETTEGREW, PAULINE K, 4601 W 109TH SUITE 225,66211
381-6765
21 F 3006 50 FP
PFUETZE, BRUCE L, 5300 W 94TH TERR,66207
381-4674
42 M 1902 68 A
PFUETZE, KARL D, 6100 MARTWAY,66202
362-7290
40 M 1902 66 CD
PHILLIPS, WARREN G, 3700 W 83RD,66208
649-0923
26 M 1902 60 P
PITTS, RONALD L, 7301 MISSION RD SUITE 249,66208
362-2524
35 M 2002 62 D
POWELL, CAROL W, 8216 CHEROKEE CIRCLE,66206
381-3785
25 F 1902 51 P
POWELL, KENNETH A, 8216 CHEROKEE CIRCLE,66206
753-7000
25 M 1902 53 IM
PRONKO, MICHAEL J, 4121 W 83RD SUITE 223,66208
648-7878
34 M 1902 60 P
RADOM, SANFORD B, PO BOX 4250,66204
677-0883
40 M 1642 66 R
REDDY, NARAYAN C, 7301 MISSION RD,66208
384-2285
45 M 49521 68 IM
REIVICH, RONALD S, 10955 GRANADA LANE,66209
383-3050
34 M 3806 60 P
RIEKE, FRANK A, 10411 WEST 55TH,66203
281-7218
16 M 1902 41 GS
ROSENBERG, STANTON L, 1900 W 75TH SUITE 200,66208
362-8080
30 M 1902 55 P
RUBIN, HERBERT M, 7301 MISSION RD,66208
362-6000
37 M 2802 63 PD

RUMOLD, MERVIN J, 6340 INDIAN LANE, 66208

03 M 1902 30 DO
SAFFO, KARL S, 10000 W 75TH ST 100,66204
362-9585
39 M 52801 62 PS
SATHYANARAYANA, SARASWATHI, 9119 W 74TH,66204
432-7419
45 F 49509 67 OBG
SAWHAR, LAXMIDAS A, 7301 MISSION RD, SUITE 312, 66208
384-4844
36 M 49523 63 IM
SCHAEFER, JOSEPH PETER, 7301 MISSION RD,66208
362-2975
34 M 1902 60 IM
SCHWEGLER, RAYMOND A, 6100 MARTWAY,66202
362-7290
37 M 1902 63 CD
SHAAD, DOROTHY J, 2322 W 51ST,66205
-
09 F 1902 44 OO
SHOFSTALL, WILLIAM H, 6100 MARTWAY,66202
722-4747
11 M 3901 41 ENT
SMITH, DALE C, 4601 W 109TH ST SUITE 224,66211
381-0353
20 M 1902 45 OPH
SMITH, DONALD J, 8600 W 95TH ST,66212
642-4515
18 M 1902 49 FP
SNODELL, FIRMIN E, 5555 W 58TH,66202
432-2080
31 M 1902 61 IM
SPEER, FREDERIC, 5811 OUTLOOK,66202
432-0625
09 M 1902 34 A
STEINER, ROBERT M, 5311 W 96TH,66207
677-0883
39 M 4117 65 R
STEINZEIG, ALFRED S, 6500 HODGES DR,66208
-
13 M 1902 37 OO
STEVENSON, E KENT, 4121 W 83RD SUITE 150,66208
649-5566
45 M 2802 67 CHP
SUGAR, ROBERT L, 4601 W 109TH,66211
341-4343
40 M 3508 66 OBG
SULLIVAN JR, HENRY B, 5817 NIEMAN RD,66203
631-6160
24 M 1902 52 FP
SUMMERVILLE, WARD W, 2120 W 49TH,66205
-
97 M 1902 27 OO
SUTTON JR, RICHARD L, 3203 W 83RD TERR,66206
-
08 M 2501 29 OO
TETZLAFF, ARCHIBALD O, 4520 W 65TH ST, 66208
334-2500
26 M 40721 52 ANES
TICE, GALEN M, 14019 W 91 TERR,66215
-
99 M 1902 29 OO
TRETBAR, LAWRENCE L, 8901 W 7TH, 66204
677-1776
33 M 1902 60 GS
TROPP, ARNOLD L, 8600 W 95TH SUITE 102,66212
648-5600
46 M 2878 72 FP
TUTERA, GINO, 11055 CEDAR,66211
341-6030
45 M 2803 71 OBG
VANNAMAN, DONALD D, P O BOX 7426,66207
381-8282
43 M 1902 71 PD
VILLANUEVA, CESAR L, 6332 ASH ST,66208
588-6200
39 M 74802 65 OBG
WANG, SIDNEY W, 9119 W 74TH ST SUITE 102,66204
722-2020
32 M 38503 58 FP
WEARE, MARY E, 4650 W 90TH TERR,66207
649-2727
48 F 4002 73 P
WEAVER, JEROME A, 8600 W 95TH SUITE 206,66212
341-4030
15 M 2802 39 PD
WEBB, JAMES R, 5949 NIEMAN ROAD,66203
631-0900
34 M 1902 61 FP

WHITEHEAD, RICHARD E, 7301 MISSION RD,66208
362-8317
31 M 2501 58 ORS
WHITLEY, DOUGLAS M, 4601 W 109TH SUITE 202,66211
341-4770
34 M 1902 60 D
WIGGINTON, GERALD D, D.O., 9119 W 74TH,66204
384-5500
44 M 2878 70 PD
WILEY, JOHN H, 9119 W 74TH ST,66204
831-2334
37 M 4113 63 OBG
WILSON, L BARRICK, 5602 FAIRWAY RD,66205
236-8423
09 M 1902 35 ANES
WILSON, ROBERT B, 7301 MISSION,66208
362-3356
10 M 1902 40 ENT
WURSTER, GEORGE R, 3700 W 83RD SUITE 203,66208
649-0923
35 M 1902 61 P
YE, RICHARD C, 7301 MISSION RD,66208
362-7505
20 M 24222 46 PS
YOHE, RUTH M, 8600 W 95TH ST,66212
383-3377
26 F 4107 54 PDA
YOUNG, JOHN W, 7301 MISSION RD SUITE 317,66208
362-7505
37 M 4706 63 PS
YOUNGSTROM, KARL A, 9660 REEDER PLACE,66214
-
08 M 3607 44 OO
ZACK, ASHLEY S, 4601 WEST 109TH SUITE 122,66211
642-4040
46 M 2803 73 PD
ZAREMSKI, SHERMAN C, 7301 MISSION RD SUITE 214,66208
831-0910
33 M 1720 58 IM
ZIMMERMAN, DANIEL D, 1900 W 47TH PL SUITE 310,66205
831-0910
45 M 3005 70 IM

SMITH CENTER—913
(Central Kansas Society)

RELIHAN, FRANCIS H, 116 SOUTH MAIN,66967
282-3291
84 M 1606 07 FP
SHEPPARD, ROBERT G, 120 E COURT,66967
282-6654
21 M 1902 45 GS
WOODS, HUGH J, 120 EAST COURT,66967
282-6654
26 M 1902 52 FP

SOUTH HAVEN—316
(Tri-County Society)

UBELAKER, ERNEST J, ,67140
892-2261
11 M 1902 38 FP

ST. FRANCIS—913
(Northwest Kansas Society)

CRAM, ERNEST R, PO BOX 625,67756
332-2511
24 M 1902 52 FP
JEWELL, ROSS L, 203 SPENCER,67756
332-2832
13 M 1902 56 FP
STEPHENSON, LUCILLE C, ,67756
-
06 F 1902 32 FP
WALZ, THOMAS J, 115 SOUTH QUINCY,67756
-
94 M 1902 21 OO

ST. JOHN—316
(Stafford County Society)

QUACKENBUSH, ROBERT P, ,67578
549-3471
21 M 1611 52 FP
SCHERER, ALFRED L, 610 E FIRST,67576
549-3261
32 M 1902 57 FP

ST. MARYS—913
(Pottawatomie County Society)

BROWN, FRED E, 602 W PALMER,66536
437-2256
26 M 1902 55 FP

STAFFORD—316
(Stafford County Society)

BROWN, C EVERETT, 102 N MAIN,67578
234-5251
10 M 1902 47 A
WARD, ROBERT L, 115 E BROADWAY,67578
234-5281
24 M 1902 52 FP

STERLING—316
(Rice County Society)

SIMPSON, TOM C, 239 N BROADWAY, 67579
278-2123
47 M 1902 73 FP

STOCKTON—913
(Central Kansas Society)

MAUCK, HAROLD C, 623 SOUTH 2ND,67669
425-6280
20 M 1902 54 FP
VOTAPKA, WILLIAM L, 623 S SECOND,67669
425-6280
24 M 1902 53 FP

SUBLETTE—316
(Southwest Kansas Society)

THIEMANN, A H, D.O., 301 OERBY,67877
675-2241
12 M 1805 42 FP

SYRACUSE—316
(Southwest Kansas Society)

PETTERSON, CECIL E, % SOUTHWEST CLINIC,67878
384-5731
14 M 1902 39 FP

TONGANOXIE—913
(Leavenworth County Society)

STEVENS, PHILIP L, BOX 319,66086
845-2090
27 M 1902 54 FP

TOPEKA—913
(Shawnee County Society)

AGAN, LAWRENCE M, 1314 PEMBROKE LN,66604
357-4306
20 M 5002 44 R
ALONSO, RENE A, CONTINENTAL MED BLDG #440,66606
357-6218
20 M 27501 46 IM
ANNAMALAI, PERIAKAN R, 1709 MEDFORD,66604
235-8600
23 M 49516 55 IM
ARREDONDO, MARIO, 6024 SW 26TH,66614
296-7216
25 M 26401 54 P
ASHLEY, BYRON J, 322 PLASS, 66611

98 M 1902 24 OO
ASHLEY JR, B JOHN, 1616 WEST 8TH ST,66606
233-2280
31 M 1902 56 OPH
AVERILL, STUART C, MENNINGER FD,66604
234-9566
24 M 502 52 P
BAEHR, RALPH H, CANCER RESEARCH CTR,66606
234-3451
35 M 1606 59 R
BAIR, GLENN O, 417 E 6TH ST,66607
354-5100
31 M 2401 57 IM
BAKER, FREDERICK C, 2101 WEST 10TH,66604
232-0909
35 M 4113 62 FP

BAKER, PHILLIP L, 300 CONT MED BLDG,66606
357-0301
37 M 3005 63 ORS
BAKER, RAY D, TOPEKA-SHAWNEE HEALTH DEP,66606
233-8961
30 M 4812 55 GPM
BARNHILL, C ALTON, MENNINGER FD,66601
234-9566
31 M 3601 56 P
BAUDE, EUGENE L ANDRE, 1815 WESTWOOD CIRCLE, 66619

02 M 39606 27 OO
BEACH, RICHARD R, 106 MED ARTS BLDG WEST,66604
233-7943
23 M 2802 48 IM
BEALE, DAVID A, MENNINGER FD,66601
234-9566
31 M 5404 56 P
BEATY, JAMES A, 1700 WEST 7TH,66606
354-1111
32 M 5101 66 EM
BECK, JOSEPH D, 2760 BURLINGAME RD,66604
235-2351
18 M 3005 43 PD
BEDFORD, D R, 6423 HUNTOON,66601

09 M 4802 40 OO
BEELMAN, FLOYD C, 1319 HUNTOON,66604

02 M 3840 35 OO
BELLER, WILLIS L, 310 MED ARTS BLDG WEST,66604
234-3451
14 M 1902 41 R
BLAKE, HENRY S, 1933 WESTWOOD DR, 66604

11 M 3520 37 OO
BOWEN, CLOVIS W, 2200 WEST 10TH,66604
234-8601
12 M 1902 37 FP
BOWEN JR, HARRY J, 2200 WEST 10TH,66604
234-8601
11 M 1902 37 FP
BRAUNSDORF, ROBERT L, 2625 OHIO,66605
354-7630
08 M 2501 35 FP
BRIDWELL, RUSSELL E, 1710 WEST 10TH,66604
234-2624
26 M 1902 51 ENT
BROOKER, ROBERT M, 314 MED ARTS BLDG EAST,66604
235-1170
18 M 1902 43 TS
BROUCEK, FRANCIS J, 2400 W 29TH,66611
266-9503
32 M 1643 58 P
CAPOOTH, L WAYNE, 2015 W 10TH,66604
235-6253
44 M 4706 70 EM
CARVER, LARRY A, 631 HORNE,66606
234-8653
37 M 1902 73 P
CASHMAN JR, MAURICE R, 901 GARFIELD,66606
354-9591
35 M 1902 61 HEM
CAVANAUGH, JOHN W, 200 PROFESSIONAL BLDG,66604
235-3488
13 M 1803 39 GS
CHAMBERLIN JR, CECIL R, BOX 829,66606
234-9566
30 M 3901 55 CHP
CHEN, TAK-MING, 303 MED ARTS BLDG,66604
234-3451
41 M 24402 68 ANES
CHERRY JR, ARTHUR C, 918 WEST 10TH,66604
233-9681
27 M 3806 53 PD
CHEUNG, P W H, 918 WEST 10TH,66604
233-9681
36 M 24338 58 R
CHOY, JAMES K L, 918 W 10TH,66604
233-9681
10 M 1643 37 U
CLARK, CRAIG N, 3124 E 6TH,66607
354-7683
29 M 1902 58 FP
COCHRAN, PAUL W, MENNINGER FD,66601
234-9566
33 M 4802 58 IM
COHEN, LOUIS, 918 W 10TH,66604
233-9681
14 M 1902 41 IM

COHEN, SIDNEY, MEMORIAL HOSPITAL,66607
354-5100
31 M 3841 57 CD
COKELEY, JOHN M, 3840 CHELMSFORD RD, 66610
272-3111
30 M 5501 55 P
COLLINS, DEAN T, MENNINGER FD,66601
234-9566
28 M 1902 55 P
COLLINS, FRANCIS T, 206 MED ARTS BLDG EAST,66604
233-6470
14 M 1902 43 IM
CONNELLY, JOHN C, MENNINGER FD,66601
234-9566
39 M 3806 65 P
CONROY, ROBERT W, MENNINGER FD,66601
234-9566
38 M 2604 64 P
COTTON, ROBERT T, 901 GARFIELD,66606
354-9591
19 M 1902 45 IM
CRARY, JOHN E, 201 MED PLAZA BLDG,66604
233-4202
18 M 1902 43 IM
CROUCH, WILLIAM H, 109 MED ARTS BLDG WEST,66604
232-8224
20 M 2802 45 PD
DEAN, DAVID B, 1808 MUNSON,66604
233-1344
39 M 4804 64 P
DELGADO, SERGIO, TOPEKA MED CENTER,66604
233-9681
37 M 2501 62 DRS
DUNAGIN, JACK A, 918 W 10TH,66604
233-9681
20 M 1902 44 P
DURST JR, ROBERT D, 1706 W 10TH,66604
357-5166
42 M 2803 69 D
ELDER, DOUGLAS M, 310 MED ARTS BLDG,66604
234-3451
41 M 1902 69 DR
FEAGAN, JERRY, 901 GARFIELD,66606
354-9591
39 M 1902 63 GE
FERNANDEZ, LUIS A, 1615 W 8TH,66604
233-8961
14 M 27501 41 PD
FIELD, RICHARD A, 303 MEDICAL ARTS BLDG,66604
235-3451
29 M 1902 55 ANES
FILLMAN, ELDON M, 301 MEDICAL PLAZA BLDG,66604
233-4256
20 M 1611 44 U
FORD, FRED L, 220 MEDICAL ARTS BLDG,66604
234-5516
11 M 2501 36 GS
FOSTER, CHARLES G, 110 CONT MED BLDG,66606
232-6964
21 M 4102 47 IM
FOSTER, D BERNARD, VA HOSPITAL,66622
272-3111
14 M 2501 38 NP
GANZARAIN, RAMON C, MENNINGER FD,66601
234-9566
23 M 23101 47 P
GAY, JOHN D, 310 MED ARTS BLDG,66604
234-3451
42 M 4802 68 DR
GENDEL, JOSEPH E, 918 W 10TH,66604
233-9681
12 M 4804 37 ORS
GLEASON, JIMMIE A, 8TH AND LINCOLN,66606
233-5101
33 M 1902 58 OBG
GOERING, EMIL L, 417 E 6TH,66607
354-5100
27 M 1902 57 IM
GOOTEE, JOSEPH E, 224 MEDICAL ARTS BLDG,66604
21 M 2834 47 DO
GRAHAM JR, CHARLES P, CON MED BLDG,66606
354-9504
40 M 3601 65 GS
GRAY, DAVID E, 115 W CRANE,66604
234-8621
16 M 1606 42 GEN
GRAYBE, ANTOINE S, SANTA FE HOSP,66607
233-2361
18 M 60501 46 IM

GREENBERG, MARK, 310 MED ARTS BLDG,66604
234-3451
46 M 1611 72 R
GREENE, HORACE T, 1710 W 10TH,66604
354-7508
15 M 401 42 FP
GREENWOOD, EDWARD D, 3617 W 6TH,66601
01 M 702 38 OO
GREER, RICHARD H, 918 W 10TH,66604
233-9681
09 M 1902 39 IM
GRIGSBY JR, GEORGE T, STORMONT VAIL HOSP,
234-9961
41 M 3601 66 IM
HAINES, ROBERT A, INSTITUTIONAL DIR,66612
296-3774
16 M 3841 42 ADM
HALLEY, M MARTIN, 40 MED ARTS BLDG,66604
233-1710
27 M 2401 53 TS
HARRIS, HUBERT L, 210 MED ARTS BLDG,66604
233-3151
12 M 1803 39 DERM
HARRIS, PATRICIA A, 1617 W 26TH,66611
354-8461
29 F 1902 54 IM
HARRISON, HALL E, 901 GARFIELD,66606
354-9591
39 M 2802 65 IM
HARTOCOLLIS, PETER, MENNINGER FD,66601
234-9566
22 M 86905 55 P
HASSELLE III, JAMES E, MENNINGER FD,66601
234-9566
35 M 4706 59 P
HEBBAR, SATYA N, 918 W 10TH,66604
233-9681
39 M 49509 62 CD
HERRERA, JORGE J, 417 E 6TH,66607
354-5100
27 M 64901 55 IM
HIEBERT, JOHN B, 420 CONT MED BLDG,66606
357-6251
40 M 1902 68 CD
HILL, ROBERT N, GARFIELD MED PARK BLDG,66606
354-9591
14 M 1902 67 IM
HIRSCHBERG, J COTTER, MENNINGER FD,66601
234-9566
15 M 1602 40 CHP
HOBBS, DONALD D, CONT MED BLDG,66606
233-7491
28 M 2401 54 ORS
HOHERZ, DAVID G, 314 MED ARTS BLDG,66604
235-1170
45 M 1902 76 TS
HORNE, JAMES B, 315 W 4TH,66603
234-2120
26 M 1606 52 P
HOYT, ARTHUR W, 2055 CLAY,66604
234-5663
14 M 2501 40 P
HSU, CHENG H, 918 W 10TH,66604
233-9681
41 M 38502 66 U
HUAMAN, ANTONIO M, 1700 W 7TH,66606
295-8060
29 M 73701 56 PATH
HUSTON, JOSEPH W, 918 W 10TH,66604
233-9681
35 M 1902 62 DRS
HUTTON, FREDERICK A, 102 MED PLAZA BLDG,66604
234-0553
29 M 6701 58 PS
JACKSON, LINDA H, 212 WOODLAWN AVE,66606
234-9566
42 F 3601 67 CHP
JACOBY II, ROBERT E, 340 CONT MED BLDG,66606
232-9394
46 M 2307 72 FP
JANSSEN, ERWIN T, MENNINGER FD,66601
234-9566
36 M 1803 62 P
JOHNSON, FREDERICK E, 1714 CENTRAL PARK,66604
92 M 2843 21 OO
JOSEPH, BRIAN W, 130 CONT MED BLDG,66606
234-8653
38 M 35205 61 CHP

JOSS, CHARLES S, 221 MED ARTS BLDG W,66604
232-0444
14 M 1606 40 FP
JOYCE, G BERNARD, 200 CON MED BLDG,66606
233-7491
17 M 1902 44 DRS
KATZ, JEROME B, MENNINGER FD,66601
234-9566
22 M 2101 44 P
KAVEL, KARL K, MED PLAZA BLDG,66604
234-2663
36 M 3605 64 PDA
KELLY, DAN A, 109 MED ARTS BLDG W,66604
232-8224
39 M 2803 64 PD
KENNEDY, HOWARD U, 1119 W 10TH,66604
233-8268
18 M 401 44 IM
KEYS JR, ROBERT C, 303 MED ARTS BLDG,66604
235-3451
36 M 1902 62 ANES
KIM, YONG W, CONT MED BLDG,66606
232-6964
28 M S8302 49 IM
KINDLING, PAUL H, 40 MED ARTS BLDG,
233-1710
30 M 3545 61 TS
KIRKEGAARD, RODGER S, 918 W 10TH,66604
233-6493
30 M 1803 56 OPH
KLEMMER, HERBERT, MENNINGER FD,66601
234-9566
11 M 1805 37 P
KOVARIK, ERNEST D, 900 WASHBURN,66604
357-5171
36 M 3005 64 OPH
KROLL, HARRY G, 200 CON MED BLDG,66606
233-7491
24 M 1602 50 ORS
LAWSON, DWIGHT, 108 MEADOW LANE,66606
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06 M 2802 30 DO
LAWSON, ROBERT C, 310 MED ARTS BLDG WEST,66604
234-3451
25 M 801 48 R
LEE, SONG DOW, 303 MED ARTS BLDG,66604
235-3451
43 M 38505 68 ANES
LEE, SONG PING, 918 W 10TH,66604
233-9681
34 M 38502 61 OTO
LENTZ, WILLIAM R, MED ARTS BLDG WEST,66604
235-3443
24 M 1902 53 FP
LESSENDEN JR, C M, 103 MEDICAL PLAZA BLDG,66604
234-5533
18 M 1902 43 D
LEVY, EDWIN Z, MENNINGER FD,66601
234-9566
29 M 1606 54 P
LONG, JOHN W, 1001 HORNE ST,66604
233-1710
43 M 1902 70 TS
LUMB, RAYMOND C, 901 GARFIELD,66606
3S4-9591
42 M 1001 68 RHU
LYNCH, JOHN A, CONT MED BLDG # 300,66606
3S7-0301
30 M 2834 55 ORS
MARSHALL, B M, 1826 SW 34TH,66611
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08 M 2802 34 U
MARTIN, WILLIAM O, 216 MED ARTS BLDG WEST,66604
235-3451
19 M 1902 44 ANES
MAU, WALTER, 301 MEDICAL PLAZA BLDG,66604
233-4256
16 M 1611 40 U
MCCARTER, DUANE K, 2101 W 10TH,66604
233-8979
26 M 1902 58 IM
MCCLELLAN, JOHN W, 1710 W 10TH,66604
234-2624
11 M 3006 36 ENT
MCCLINTOCK, EDWARD A, III MEDICAL ARTS BLDG,66604
3S4-8518
07 M 1902 33 IM
MCCLURE, JAMES A, 301 MED PLAZA BLDG,66604
233-4256
18 M 1902 44 U

MCELROY, ROBERT T, 221 MED ARTS BLDG,66604
232-0444
35 M 1902 61 GS
MEGIBOW, ALAN D, 918 W 10TH,66604
233-9681
37 M 3503 64 CHP
MEIDEINGER, RICHARD, 310 MED ARTS BLDG,66604
234-3451
39 M 1902 65 DR
MENNINGER, KARL A, BOX 829,66601
234-9566
93 M 2401 17 P
MENNINGER, ROBERT G, 3617 W 6TH ST,66601
234-9566
22 M 3545 52 P
MENNINGER, ROY W, 3617 W 6TH,66601
234-9566
26 M 3520 51 P
MENNINGER, W WALTER, THE MENNINGER FD,66601
234-9566
31 M 3520 57 P
MILLS JR, PHILIP E, 901 GARFIELD,66606
3S7-6171
36 M 1902 64 N
MITTLEMAN, FREDERICK S, MENNINGER FD,66601
234-9566
45 M 3006 70 P
MODLIN, HERBERT C, MENNINGER FD,66601
234-9566
13 M 3005 38 P
MOORE, HUGH C, 1500 WEST 10TH,66604
234-9961
33 M 4812 59 PATH
MORAN, JEFFERY, MENNINGER FD,66601
234-9566
47 M 42901 72 P
MORRIS, MERLE O, 1401 W 10TH,66604
234-2877
21 M 1902 45 OBG
MORROW JR, J TARLTON, MENNINGER FD,66601
234-9566
23 M 4804 47 CHP
MOYER, JOHN D, 301 MEDICAL PLAZA BLDG,66604
233-4256
39 M 1902 65 U
MUELLER, ARNOLD V, 918 WEST 10TH,66604
233-9681
31 M 3005 57 IM
MYERS, JO ANN, MENNINGER FD,66601
234-9566
28 F 1902 53 P
NABOURS, RICHARD D, 4228 W 29TH ST TERR,66614
272-7190
27 M 1902 54 FP
NELSON, JAMES N, 918 W 10TH ST,66604
233-9681
30 M 1902 59 P
NICE, G WILLIAM, 112 MED ARTS BLDG EAST,66604
235-8090
22 M 1902 46 IM
NOVOTNY, PETER C, MENNINGER FD,66606
234-9566
30 M 15407 55 P
NYSTROM, CURTIS A, 2707 W 29TH,66614
272-8440
23 M 1902 54 FP
OBOURN, ROBERT L, 1150 OAKLEY,66604
234-9566
19 M 2802 50 P
OLMO TERRON, NEFTALI, MENNINGER FD,66601
234-9566
38 M 84705 65 P
ONEIL, ROBERT H, 901 GARFIELD,66606
3S4-9591
20 M 1902 45 IM
PARMAN, ROBERT D, 109 MED ARTS BLDG WEST,66604
232-8224
27 M 1902 54 PD
PATEL, VINOD, 901 GARFIELD,66606
3S7-6171
47 M 49531 69 N
PATINO, EDGAR, MENNINGER FD,66601
234-9566
41 M 26406 66 P
PAYNE, ROBERT R, 200 CONTINENTAL MED BLDG,66606
233-7491
29 M 1902 55 ORS
PENN, GEORGE M, MENNINGER FD,66601
234-9566
30 M 4802 58 P

PETERSON, DEAN L. 303 MED ARTS BLDG WEST.66604
235-3451
24 M 1902 54 ANES
PETERSON, ROBERT L. 1500 W 10TH.66604
342-2341
36 M 1902 62 FP
PETERSON, VERNON J. 310 MED ARTS BLDG.66604
234-3451
42 M 512 68 R
PETRIK, EDWIN L. MEMORIAL HOSPITAL.66607
354-5100
35 M 1902 64 HEM
PFUETZE, ROBERT E. 209 MED ARTS BLDG EAST.66604
232-9257
09 M 1902 35 OBG
PIERCE, CHARLES F. 918 W 10TH.66604
233-9681
24 M 4101 91 OBG
PIERCE, DONALD R. 307 MED ARTS BLDG EAST.66604
235-2226
23 M 5101 49 FP
POGGI, RAYMOND G. MENNINGER FD.66601
234-9566
33 M 2604 68 P
POLLY, RICHARD E. 631 HORNE ST.66606
357-0301
42 M 1803 68 ORS
PORTER, ROBERT D. 901 GARFIELD.66606
354-9591
41 M 2802 67 IM
POWELL, WILLIAM R. 833 GARFIELD.66606
233-8941
30 M 1902 54 GS
POWELL II, BENSON M. 400 CONTINENTAL MED BLDG.66606
354-9504
26 M 1606 49 TS
PRESTON, RALPH R. 1710 WEST 10TH.66604
234-2624
19 M 1902 44 OPH
PROKOP, BRADFORD S. 900 WASHBURN.66604
357-5171
32 M 1606 57 OPH
PYLE, LUCIEN R. 211 MED ARTS BLDG EAST.66604

01 M 1601 28 OO
RAINBOW, KATHRYN A. TOPEKA STATE HOSPITAL.66606
296-4503
21 F 4707 48 P
RAMSEY, BARTLETT W. 1001 HORNE ST SUITE 205.66604
234-3465
25 M 1902 50 PD
RANSDELL, EDGAR C. 8TH & LINCOLN.66606
233-5101
41 M 3005 66 OBG
RANSOM, JAMES H. 223 MEDICAL PLAZA BLDG.66604
234-2663
36 M 1803 62 A
REIMER, DONALD R. ANES DEPT ST FRANCIS HOSP.66606
354-8411
34 M 702 62 ANES
REINKING, VICTOR E. 918 WEST 10TH.66604
233-9681
26 M 1902 51 IM
RENDON, HUMBERTO M. 115 W CRANE.66603
234-8621
36 M 73705 64 PATH
REYMOND, RALPH D. 310 MED ARTS BLDG.66604
234-3451
37 M 2301 67 R
RICHARDSON, J M. 918 W 10TH.66604
-
M 1611 IM
RIEDEL, ROBERT H. 6731 SW ALYESBURY RD.66610
-
04 M 2802 28 OO
ROBERTS, HOWARD E. 700 HARRISON.66603
354-8461
12 M 2105 40 ADM
ROBERTS, WARREN E. PO BOX 4047.66601
272-5797
25 M 1902 57 FP
ROBINSON, DAVID B. 800 LINCOLN.66606
233-5101
47 M 1902 73 OBG
ROCERETO, PAUL V. 1001 MULVANE.66604
233-0401
47 M 1902 73 FP
ROEDER, ROBERT E. 901 GARFIELD.66604
S34-9591
40 M 1902 67 IM

ROSKOS, S RICHARD, MENNINGER FD.66601
234-9566
42 M 2604 68 P
ROSS, JACK L. MENNINGER FD.66601
234-9566
32 M 4812 56 P
ROVINSKI, HELEN T. 2200 GAGE BLVD.66614
272-3111
16 F 1001 43 PM
ROY, WILLIAM R. 1700 W 7TH.66606
295-8000
26 M 1606 48 ADM
RUCKER, CLEMENS. 1205 W 29TH.66611
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86 M 2822 05 OO
RUNNELS, JOHN B. 901 GARFIELD.66606
357-6171
35 M 1902 61 NS
RUPP, RICHARD J. 901 GARFIELD.66606
354-9591
42 M 3841 68 CD
SANCHEZ, ROGELIO. 918 WEST 10TH.66604
233-9681
31 M 64901 64 U
SARGENT, JOSEPH D. MENNINGER FD.66601
234-9566
32 M 2501 58 IM
SAYLOR, EDWARD H. 918 WEST 10TH.66604
233-9681
39 M 1902 65 PD
SAYLOR, LESLIE L. 918 WEST 10TH.66604
233-9681
07 M 1606 35 GS
SAYLOR, MARK. 918 WEST 10TH.66604
233-9681
37 M 1902 66 GS
SCAMMAN, W WIKE. 115 W CRANE.66603
234-8625
32 M 4705 57 PATH
SCHLOESSER, HARVEY L. 918 MERCHANTS NATL BK.66612
235-3184
21 M 3901 51 P
SEGERSON, JOHN A. 901 GARFIELD.66606
357-6171
18 M 3545 43 N
SEHDEV, JOAN. MEMORIAL HOSP.66607
354-5100
40 F 6101 63 FP
SETTLE SR, RUSSELL O. 2320 CHELSEA DRIVE.66614

04 M 1902 29 OO
SEVIER, SAMUEL M. 3107 W 21ST.66604
296-5306
19 M 4812 44 IM
SHAW, JOSEPH L. MED ARTS BLDG.66604
235-6221
34 M 511 60 ORS
SHEAFOR, DOUGLAS. 918 W 10TH.66604
233-9681
34 M 1902 60 P
SHELTON, STEPHEN E. 918 W 10TH.66604
233-9681
35 M 702 61 P
SHERWOOD JR, CLARENCE E. CONTINENTAL MED BLDG.66606
354-9504
22 M 702 53 GS
SIMPSON, WILLIAM S. MENNINGER FOUNDATION.66601
234-9566
24 M 6001 48 P
SISK, PHILLIP B. 310 MED ARTS BLDG WEST.66604
234-3451
32 M 1803 56 R
SMITH, LEO A. 918 BUCHANAN.66604
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08 M 3006 33 OO
SPEARMAN, JESSE L. MED ARTS BLDG ROOM 24.66604
234-2879
20 M 1902 54 OBG
SPENCER, MILLARD C. 310 MED ARTS BUILDING.66604
234-3451
28 M 1902 55 R
SPENCER, WAYNE E. 103 MED ARTS BLDG EAST.66604
233-9686
38 M 1902 64 IM
STEIN, JOSEPH M. 901 GARFIELD.66606
357-6171
24 M 3519 47 N

STOCK, KARL W, 1710 W 10TH,66604
235-5205
13 M 2834 37 OPH
SUTTON, RICHARD O, 200 CONT MED BLDG,66606
233-7491
38 M 4706 67 ORS
SWOGGER JR, GLENN, MENNINGER FD,66601
234-9566
35 M 3806 60 P
TAKAHASHI, TETSURO, MENNINGER FD,66614
234-9566
32 M 57211 60 P
TAPPEN, DANIEL L, 8TH AT LINCOLN,66606
233-5101
16 M 1902 41 OBG
TARGDWNIK, KARL K, K-STATE REC & DIAGNOSTIC,
296-7281
15 M 40710 49 P
TARNDWER, WILLIAM, MENNINGER CLINIC,66601
234-9566
21 M 4802 48 P
TEMPERO, STEPHEN J, 310 MED ARTS BLDG,66604
234-3451
42 M 1606 67 R
TEUSINK, J PAUL, MENNINGER FD,66601
234-9566
41 M 2501 69 P
THOMS, NORMAN W, 40 MED ARTS BLDG,66604
233-1710
34 M 2501 59 TS
THURSTON, DAVID E, 200 CONTINENTAL MED BLDG,66606
233-7491
29 M 1902 55 ORS
TOZER, RICHARD C, 901 GARFIELD,66606
357-6171
19 M 4102 45 NS
TRAVIS, JOHN W, CANCER RESEARCH CENTER,66606
295-8000
29 M 1606 55 R
TREES, CLYDE B, 3700 HUNTOON,66604
09 M 2401 33 OO
TREGER, NEWMAN V, 1704 W 10TH,66604
354-8761
16 M 1902 40 IM
*TRUITT, WILLIAM ROY, 1700 W 7TH,66606
354-8411
37 M 3515 66 EM
UHR, NATHANIEL, MENNINGER FOUNDATION,66601
00 M 3519 21 OO
VANDE GARDE, LARRY D, 800 LINCOLN,66606
233-5101
41 M 1803 66 OBG
VISWANATHAN, BYRAVAN, 417 E 6TH,66614
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39 M 49534 62 HEM
VOTH, HAROLD M, MENNINGER FOUNDATION,66601
234-9566
22 M 1902 47 P
WALLACE, LEO F, 1700 W 7TH,66614
17 M 1902 41 EM
WALLS, WILLIAM J, 310 MED ARTS BLDG,66604
234-3451
39 M 2834 66 DR
WALZ, ROYCE C, 900 WASHBURN,66610
234-2676
27 M 15407 60 P
WARD, HOWARD N, 901 GARFIELD,66606
354-9591
37 M 1606 62 HEM
WARE, LUCILE M, MENNINGER FOUNDATION,66601
234-9566
29 F 3501 53 P
WEAVER, WALTER D, 900 WASHBURN ST,66606
357-5171
41 M 1902 69 OPH
WEBER, DARRELL J, 1710 W 10TH,66604
233-2305
16 M 1902 44 FP
WHITE, HARRY H, 901 GARFIELD,66606
357-6171
34 M 1902 58 N
WIKSTEN, VERNON C, 807 TERRACE AVE,66611
11 M 1902 37 OO
WILCOX, DONALD E, 6700 S TOPEKA AVE,66606
296-3782
24 M 1902 55 PH

WILLIAMS, HOWARD V, STATE OFFICE BUILDING,66612
296-3471
18 M 1205 43 P
WILSON, JAMES W, KS DEPT OF HEALTH,66622
862-9360
26 M 3901 58 PATH
WILSON, MARVIN H, 38 MED ARTS BUILDING,66604
234-0591
38 M 1003 64 S
WING, NANCY J, 700 HARRISON,66603
295-3175
30 F 5104 56 ADM
WONG, NORMUND, MENNINGER FD,66601
234-9566
34 M 502 59 P
WOODS, ROBERT P, 901 GARFIELD,66606
357-6171
14 M 6701 40 N
WOOLLCOTT JR, PHILIP, MENNINGER CLINIC,66601
234-9566
28 M 4102 54 P
YOUNG, THEODORE E, 107 MED ARTS BLDG WEST,66604
232-0576
22 M 2307 46 PD
ZIMMERMAN, WILLIAM H, 515 MILLS BUILDING,66612
232-4377
20 M 3006 52 S

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WERNER, WILLARD F, ,67879
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24 M 1902 52 FP

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356-1261
19 M 1902 46 FP
TILLOTSON, DON R, 223 N MAIN,67880
356-1261
32 M 1902 65 FP

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DANIELS, ROBERT M, 130 MILES AVE,67147
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24 M 1902 54 FP
WILSON, ROBERT L, RR 1,67147
685-2563
30 M 1902 57 EM

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BERNER, NEAL E, 200 N 6TH,67672
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44 M 1902 72 FP
CLARK, DAVID H, MEDICAL CENTER BLDG,67672
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36 M 1902 62 FP
HAMILTON, JAMES J, MEDICAL CENTER,67672
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30 M 1902 55 FP

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29 M 1902 60 FP
BRADEN, BILL L, 507 ELM,66547
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31 M 1902 60 FP
CLARK, LAURENCE A, 507 ELM,66547
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12 M 1902 42 FP

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WARREN, LINDA D, 103 E 3RD,66968
325-2240
44 F 1902 70 FP

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PETERSON JR, EVAN A, 324 ST JOSEPH ST,66090
989-3122
24 M 1803 55 FP

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ANDERSON, LARRY R, 1323 NORTH A,67152
326-3301
43 M 1902 73 FP
COLE, WARD M, 110 N JEFFERSON,67152
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08 M 1902 36 FP
DIACON, JAMES L, 124 E LINCOLN,67152
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24 M 3901 52 FP
HESSE, ROBERT S, 1323 NORTH A,67152
326-7792
43 M 1902 69 PATH
MCCORMICK, EUGENE C, SECURITY STATE BANK BLDG,67152
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31 M 1902 56 FP
MCGOVERN, JAMES L, 124 E LINCOLN,67152
326-2111
22 M 3901 52 FP
NIEBLES, ANGEL, 1322 N A,67152
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WEIGAND, JOEL T, 1323 NORTH A,67152
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43 M 1902 70 FP

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SIMMONS, WILLIAM C, 612 MAIN,66092
883-2191
34 M 1902 60 FP

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DECHAIRO, THOMAS, DECHAIRO HOSP,66549
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13 M 1902 36 FP

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ADAMS, AUSTIN J, 435 N ROOSEVELT,67208
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10 M 4804 36 OD
AGUSTIN, CONRADO M, 1035 N EMPORIA,67214
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38 M 74807 62 OBG
AHLSTRAND, RICHARD A, 3333 E CENTRAL,67208
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41 M 3005 67 R
ANDERSON, DONALD S, 3333 E CENTRAL,67208
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42 M 1902 69 ANES
ANDERSON, EUGENE G, SUTTON PLACE,67202
265-8619
19 M 1902 44 OBG
ANDERSON, HARRY D, 14516 E PAWNEE,67230
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08 M 1601 34 OD
ANDERSON, V GARY, 1520 S CLIFTON,67218
685-2371
42 M 3901 67 EM
ARTZ, TYRONE D, 3244 E DOUGLAS,67208
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41 M 1803 67 ORS
ASHMORE, ARTHUR L, 5025 E KELLOGG,67218

05 M 1902 32 OD
AUNINS, JOHN, 4853 HEMLOCK,66048
524-6805
28 M 4706 56 FP
BAILEY, DONALD C, 3243 E MURDOCK,67208
685-1491
37 M 3901 65 ORS
BAIR, ALBERT E, 14504 WILLOWBEND CT,67230
689-9163
16 M 1902 44 GS
BARBA, ANTONIO P, 1035 NORTH EMPORIA #165,67214
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34 M 74807 62 OBG
BARBA, ESTRELLA G, 1035 NORTH EMPORIA,67214
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BARKER, BENJAMIN W, 1148 S HILLSIDE,67211
683-4647
18 M 1902 51 FP
BARNETT, ARNOLD M, 3243 E MURDOCK,67208
685-2377
32 M 83601 54 N
BARTLETT, WAYNE C, 3244 E DOUGLAS,67208
689-9124
07 M 1601 31 OD
BASS II, ORAL E, 851 N HILLSIDE,67214
685-1371
40 M 2803 71 U
BATES, MICHAEL D, 2703 EAST CENTRAL,67214
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48 M 3005 74 OBG
BAUMAN, M LEON, 1629 UNIVERSITY,67213
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01 M 1902 44 OD
BAUMANN, PAUL A, 3333 E CENTRAL,67208
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32 M 5605 57 R
BEAVER, JAMES L, 959 N EMPORIA,67214
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09 M 2101 35 GS
BEBAK, DONALD M, 2322 E CENTRAL,67208
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32 M 3508 58 ANES
BECKER, KARL EDMUND, 3243 E MURDOCK ST 101,67208
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43 M 2307 69 A
BELL, JOHN D, 1035 NORTH EMPORIA #105,67214
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BETHEL, CHANDLER S, 5107 E 21ST ST,67208
682-6559
34 M 1902 59 IM
BIERMANN, HENRY J, 425 E MURDOCK,67214
265-6287
27 M 3006 52 GS
BIERMANN, WILLIAM J, 1435 LIEUNETT,67203
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04 M 3006 29 OD
BINYON, KERNIE W, 4618 E CENTRAL,67208
684-2819
24 M 1902 56 FP
BLAYLOCK, HOYT C, 835 N HILLSIDE,67214
685-4395
21 M 1902 45 D
BLOOD, MARY J, 647 N HILLSIDE,67214
682-4559
13 F 1902 49 PD
BOLT, MICHAEL, 424 NORTH YALE,67208
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BOND, ROGER C, 3243 E MURDOCK,67208
684-0251
40 M 5606 67 CD
BOSILJEVAC, JOSEPH E, 550 NORTH HILLSIDE,67214
-
BOWMAN, HAROLD S, 1441 NORTH ROCK RD,67206
-
04 M 2802 30 OD
BOYD, Z REX, 120 S MAIZE RD #12,67209
262-6211
26 M 3005 52 FP
BOYLE, HUGH H, 424 N WOODLAWN,67208
686-2193
33 M 3806 60 PATH
BRAKE, DAVID, 3333 E CENTRAL,67208
685-1291
43 M 702 68 R
BRAUN, THOMAS G, 3243 NORTH MURDOCK #601,67208
685-2377
35 M 6001 61 N
BRAUN III, WILLIAM T, 3333 E CENTRAL,67208
685-1291
37 M 2802 61 R
BRECKBILL, DAVID L, 3333 EAST CENTRAL #214,67208
-
R

BRINTON, EDWARD S. 329 NORTH TERRACE DR.67208
262-6211
15 M 1611 41 OO
BRITO, RAUL E. 3243 E MURDOCK.67208
682-4523
32 M 31901 59 U
BROSIUS, FRANK C. 3243 E MURDOCK.67208
684-0251
25 M 1902 49 IM
BROWN, DAVID J. 425 EAST MURDOCK.67214
265-6287
45 M 1902 71 GS
BROWN, ROBERT L. 5025 E KELLOGG.67218
682-1534
21 M 1902 49 FP
BROWN, RONALD C. 3243 E MURDOCK.67208
685-8231
47 M 2803 73 FP
BROWN, RONALD L. 303 S HILLSIDE.67211
684-7251
45 M 3901 71 ANES
BROWN, VAL J. 1802 N HYDRAULIC.67214
265-1461
24 M 1003 47 FP
BROWNING, WILLIAM H. 851 N HILLSIDE.67214
685-1371
16 M 1902 43 U
BRUNER JR, KENNETH W. ST FRANCIS HOSPITAL.67214
262-6211
44 M 2401 70 PATH
BUBECK, RALPH W. 3244 E DOUGLAS.67208
689-9396
36 M 1803 62 IM
BUCK JR, BEN H. 510S E 21ST.67208
684-2081
17 M 2834 43 TS
BUOETTI, JOSEPH A. 427 N HILLSIDE.67214
686-6608
11 M 3509 36 ENT
BURNEY, WILLIAM W. 1755 N MADISON.67214
264-8311
17 M 1902 52 FP
BURPEE, JAMES F. 851 N HILLSIDE.67214
685-1371
39 M S605 66 U
BUTH, DENNIS K. 2916 EAST CENTRAL.67214
684-5243
45 M 1902 72 IM
BUTIN, J WALKER. 3244 E DOUGLAS.67208
689-9477
23 M 1902 47 IM
CALIENDO JR, DANIEL J. WESLEY MED CENTER.67214
685-2563
41 M 1902 67 EM
CAPPER, STANLEY L. 3244 E DOUGLAS.67208
689-9206
37 M 1803 67 D
CARNAHAN, ROBERT L. 3244 EAST DOUGLAS.67208
689-9325
42 M 1902 70 IM
CARREAU, ERNEST P. 5105 E 21ST.67208
682-0281
17 M 1902 44 GS
CARTER, MACK A. 3333 E CENTRAL.67208
684-5158
18 M 1902 50 OPH
CARTWRIGHT, LYLE B. 1070S RINGER.67209
722-2148
37 M 3901 65 OM
CASEY, JOHN J. 3243 E MURDOCK.67208
685-2377
25 M 2604 49 NS
CAUBLE, WILBUR G. 1148 S HILLSIDE.67211
683-1681
12 M 2834 39 GS
CAWLEY, LEO P. WESLEY MED CENTER.67214
685-2151
22 M 3901 52 PATH
CHANG, FREDERIC C. 905 N EMPORIA.67214
263-0296
35 M 2401 59 GS
CHAPMAN, JAMES H. POST OFFICE BOX 11344.67211
265-1684
27 M 4706 63 R
CHARD, FREDERICK H. 3244 E DOUGLAS.67208
689-9129
15 M 5605 39 D
CHIU, CHU-SHIN. 3244 E DOUGLAS.67208
689-9319
38 M 2604 66 IM

CHO, SECHIN. 1001 NORTH MINNEAPOLIS.67214
47 M S8302 71 PO
CHRISTMAN, CARL G. 345 N HILLSIDE.67214
683-7539
48 M 4802 74 OBG
CLARK, COURTNEY. 303 S HILLSIDE.67218
684-7251
30 M 1902 56 ANES
CLEAVER, EDGAR M. 1900 EAST 9TH.67214
268-8391
26 3005 54 PH
CLIFTON, H DAVID. 3400 EAST GRAND.67218
685-1111
41 M 401 65 R
CLYDE, HARRIE R. 3244 EAST DOUGLAS.67208
689-9185
41 M 1902 67 U
* COHEN, JUSTIN T, 3333 E CENTRAL, 67208
684-5158
47 M 2803 74 OPH
COHLMIA, JERRY B. 103S N EMPORIA.67214
263-5891
43 M 1902 70 IM
COLEMAN, THOMAS J. 959 N EMPORIA.67214
265-0749
18 M 3545 51 IM
COLLIER, HAROLD W. 303 SOUTH HILLSIDE.67211
684-7251
45 M 1902 71 ANES
CONCEPCION JR, EUGENIO S. 1152 SOUTH CLIFTON.67218
681-2401
39 M 74802 64 CO
CONRARDY, PETER A. 3243 E MURDOCK.67208
686-7327
42 M S02 69 ANES
COOK, G EDWARD. ST JOSEPH HOSPITAL.67218
685-1111
42 M 401 67 R
COOK, O RAY. 315 N HILLSIDE.67214
686-3391
42 M 2012 71 FP
CORRIGAN, GEORGE F. 711 STACKMAN DRIVE.67203
95 M 3006 20 OO
COSSMAN, F PRICE. 951 N HILLSIDE.67214
685-1371
28 M 1902 57 U
COWLES, GEORGE E. 47 MISSION RD.67206
96 M 1902 21 OO
COWLES, GORDON T. 3333 E CENTRAL.67208
683-2661
32 M 1902 58 OBG
CRANE, DAVID D. 929 N ST FRANCIS.67218
262-6211
34 M 2501 60 PATH
CRISWELL, MILDRED LOUISE. 1520 SOUTH CLIFTON.67218
685-2371
42 F 1902 67 EM
CRONIN, DONALD J. 3244 E DOUGLAS.67208
689-9227
16 M 2604 40 ENT
CROW, ERNEST W. 3243 E MURDOCK.67208
684-0252
20 M 1902 44 CD
CROWLEY, EDWARD X. 345 N HILLSIDE.67214
682-4519
14 M 1643 39 GYN
CUMMINGS, RICHARD J. 427 N HILLSIDE.67214
686-6608
32 M 1902 57 OTO
DAVENPORT, S SCOTT. 3333 E CENTRAL.67208
684-8811
30 M 401 61 N
DAVIDSON, HARRY T. 556 N BROADVIEW.67208
87 M 3802 11 OO
*DAVIS, PAUL H. 315 N HILLSIDE, 67214
685-8231
47 M 3901 72
DAVIS, RONALD B. 1148 S HILLSIDE.67211
685-2152
46 M 1902 72 FP
DE BAKKER, JAN B. 1035 N EMPORIA.67214
263-4903
25 M 5104 59 GS
DECKERT, ROSALIE E. 3244 EAST DOUGLAS.67208
689-9212
47 F 2803 73 IM

DEJONG, DAVID C, ST FRANCIS HOSPITAL,67214
262-6211
33 M 2501 59 PATH
DEMOSS, ELEANOR P, 3333 EAST CENTRAL #201,67208
682-5523
42 F 74802 66 PD
DONNELL, JAMES M, 3306 E CENTRAL,67208
682-6121
28 M 1902 55 FP
DOORNBOS, J FRED, 929 N ST FRANCIS,67214
262-6211
28 M 1902 57 R
DRAKE, RALPH L, 4422 E 3RD,67208
-
99 M 4102 26 OO
DREVETS, CURTIS C, 3244 E DOUGLAS,67208
689-9178
30 M 1902 56 IM
DRIGGS, GUY K, 929 NORTH ST FRANCIS,67214
-

DUICK, GREGORY, 1035 NORTH EMPORIA #130,67214
263-3271
46 1643 72 CO
DURAND, ANTONIO C, 959 N EMPORIA,67214
263-7893
29 M 74807 56 U
DWORZAK, DAVID L, 1001 N MINNEAPOLIS, 67214
262-6211
48 M 1902 73 IM
DYCK, CORA E, 702 WAVERLY,67218
-
00 F 1902 26 OO
DYCK, GEORGE, 1001 N MINNEAPOLIS,67219
268-8388
37 M 06201 64 P
DYER, VERNON E, 3244 EAST DOUGLAS,67208
689-9234
36 M 301 72 OBG
ECKERT, WILLIAM G, ST FRANCIS LAB,67214
262-6211
26 M 3519 52 PATH
EDWARDS, MANIS C, 3333 E CENTRAL,67208
683-2661
33 M 3005 58 OBG
EGELHOF, RICHARD H, 841 N BROADWAY,67214
263-6131
45 M 1902 73 FP
ELLIS, HARVEY O, 6611 E CENTRAL,67206
683-1022
24 M 1902 55 GS
ELNEN, WALTER T, 460 N TERR DR,67208

03 M 1643 32 OO
ELNESER, RAFAEL, 929 NORTH ST FRANCIS,67214
262-6211
39 M 84708 67 OBG
EMERY, FRANK A, 10 ST JAMES PLACE,67206
-
05 M 2802 29 OO
ERKEN, RONALD V, 3101 E 9TH,67214
684-0201
29 M 2834 56 P
EVANS, FARRIS D, 521 RUTLAND RD,67206

05 M 1902 32 OO
EVANS, GRANT E, 3333 E CENTRAL,67208
682-6556
21 M 4901 46 OBG
EVANS, KENNETH L, 1131 SOUTH CLIFTON,67218
685-4354
42 M 3901 67 FP
FARHA, GEORGE J, 905 N EMPORIA,67214
263-0296
27 M 2101 57 GS
FARHA, S JIM, 905 N EMPORIA,67214
263-0296
31 M 1001 57 TS
FENDER JR, THOMAS H, 925 N EMPORIA,67214
267-8439
25 M 4812 54 NP
FERRIS, BRUCE G, 825 N EMPORIA,67214
262-3495
43 M 1902 69 PS
FEUILLE, JR, EDMOND G, 345 N HILLSIDE, 67214
682-4572
50 M 4802 75 OBG
FINLEY, DENNIS R, 1035 N EMPORIA,67214
262-7429
36 M 1606 62 ORS

FIRKINS, RICHARD T, 1035 N EMPORIA,67214
263-5865
31 M 1803 56 ENT
FISHER, JAMES B, 3244 E DOUGLAS,67208
689-9477
09 M 1902 36 GE
FITZGERALD, EDWARD J, 3400 GRAND,67218
685-1111
22 M 3006 50 R
FLEMING, FORNEY W, 3243 EAST MURDOCK #200,67208
685-1491
43 M 4802 69 ORS
FLOWERS JR, CLELL B, 855 N HILLSIDE,67214
685-1381
22 M 1902 55 FP
FORD, CHARLES R, 1301 N WEST ST,67203
942-4279
38 M 1902 63 OPH
FOWLER, ROBERT J, 3244 E DOUGLAS,67208
689-9236
37 M 2802 63 IM
FRANCIS, NORTON L, 5205 E 21ST,67208
684-2838
10 M 3005 35 ENT
FRITZ, GEORGE E, WESLEY MED CENTER,67214
685-2151
18 M 2507 43 PATH
FRITZEMEIER, WILLIAM H, 835 N HILLSIDE,67214
685-4395
14 M 1902 41 O
FROMM, ARTHUR H, 315 N HILLSIDE,67214
685-2281
37 M 1902 63 FP
FULTON, JOHN K, 3333 E CENTRAL,67208
686-0732
18 M 5605 43 PUD
GAINES, LARRY S, 3333 E CENTRAL, 67208
685-1219
40 M 4501 66 TR
GALICHIA, JOSEPH P, 1035 NORTH EMPORIA #130,67201
263-3271
42 M 1902 69 CO
GALVAN, ALONSO, 3243 E MURDOCK,67208
684-0251
38 M 64906 64 IM
GEMILO, AMANCIO C, 1520 SOUTH CLIFTON,67218
685-2371
37 M 74801 61 EM
GENILO, CELESTE A, 3244 EAST DOUGLAS,67208
689-9445
39 F 74801 62 A
GEORGE, EARL F, 406 E CENTRAL,67202
265-6991
35 M 1902 65 FP
GEORGE, M DON, 3101 E NINTH,67214
268-8388
31 M 1001 56 P
GESSLER, DONALD J, 1122 N TOPEKA,67214
265-2876
41 M 1902 67 FP
GILMARTIN, RICHARD C, 3243 EAST MURDOCK #601,67208
685-2377
32 M 4101 58 CHN
GIVNER, DAVID, 611 N HILLSIDE,67214
684-0501
03 M 2301 29 IM
GOERING, ROBERT C, 3400 GRAND,67218
685-1111
25 M 64901 56 PATH
GOLD, JAY H, 550 NORTH HILLSIDE,67214
685-2151
47 M 2401 73 PATH
GOLDBERG, HERBERT R, 400 N WOODLAWN,67208
685-4215
33 M 3508 59 PD
GONZALEZ, FRANCISCO, 1035 NORTH EMPORIA #265,67214
265-3226
46 M 73706 70 IM
GONZALEZ, HIRAM, 1035 N EMPORIA,67214
262-1853
20 M 64901 52 P
GOODIN, ETHELYNN A, 3244 EAST DOUGLAS,67208
689-9332
42 F 401 66 P
GOODIN, WALKER D, 3244 EAST DOUGLAS,67208
689-9332
34 M 1720 62 P
GOODPASTURE, HEWITT C, 1001 N MINNEAPOLIS,67214
262-6211
43 M 1902 69 IM

GOULONER, RENE M. 139 COURTLEIGH DR.67218
-
88 M 3501 18 OO
GOYLE, KRISHAN K. 1111 N ST FRANCIS.67206
265-2613
34 M 49529 63 IM
GOYLE, VIMAL. 1111 N ST FRANCIS.67206
265-2316
41 F 49529 6S OBG
GRAUEL, CHARLES W. 8310 CHALET.67208
262-1127
44 M 1902 70 ANES
GRAUEL, TRUMAN W. 3333 E CENTRAL.67208
682-6S11
41 M S605 66 OBG
GRAVES, JACK W. 3244 E DOUGLAS.67208
689-9124
17 M 1902 42 GS
GRAY, C LUCIEN. 4821 E CENTRAL.67208
684-5171
21 M 1902 45 ENT
GRAY, H TOM. 3244 E DOUGLAS.67208
689-9152
19 M 401 44 D
*GREER, JAMES A. 3244 E MURDOCK.67206
689-9227
43 M 1611 69 OTO
GRIBBLE, ROBERT N. 925 N ST FRANCIS.67211
262-6211
43 M 1902 69 R
GRILLOT, FLOYD B. 814 S WOODLAWN.67218
684-0243
18 M 1902 S1 FP
GROHS, HEINZ K. 550 NORTH HILLSIDE.67214
68S-21S1
42 M 15407 66 PATH
GRUNO, FRANK M. 3244 EAST DOUGLAS.67208
689-9420
47 M 1803 72 IM
GRUSHNYS, ARNOLD. 3244 E DOUGLAS.67206
689-944S
19 M 40721 S9 ANES
GSELL, GEORGE F. 3244 E DOUGLAS.67208
689-9316
07 M 1601 33 OPH
GUTHRIE, RICHARD A. 1001 N MINNEAPOLIS.67214
268-8228
35 M 2803 60 PO
HAGAN, C THOMAS. 959 N EMPORIA.67214
265-0789
16 M 3006 42 IM
HAGAN, FRANCIS J. PO BOX 1837.67201
262-1057
13 M 3006 39 FP
HALE, RALPH. 847 S HILLSIDE.67211
684-0295
18 M 1902 46 A
HALPIN, EDWARD O. 1148 S HILLSIDE.67211
685-9229
33 M 1902 62 OBG
HARMS, EDWIN M. 5623 POLO DR.67208
-
06 M 3901 34 OO
HARRIS, FRANK H. 1035 N EMPORIA.67214
262-18S3
09 M 1001 39 NP
HARRISON, A BROOKS. 3333 E CENTRAL.67208
682-2314
99 M 1902 25 OO
HART, DILLIS L. 510S E 21ST.67208
684-2081
36 M 3901 64 GS
HARVEY, ROSEMARY B. 1801 E 10TH.67214
265-5674
24 F 1902 49 ADM
HARVEY, THOMAS S. 2007 SOUTH HYDRAULIC.67211
267-8247
12 M 801 41 PATH
HATTRUP, RICHARD J. 610 N TYLER RD.67212
681-3016
31 M 3006 57 FP
HAWLEY, RAYMOND G. ST JOSEPH HOSP.67218
685-1111
39 M 1902 65 PATH
HAYES, WILLIAM L. 3243 E MURDOCK.67208
684-02S1
28 M 1902 53 CO
HAYS, THOMAS H. 31S NORTH HILLSIDE.67206
681-1841
49 M 1902 7S FP

HENNING, CHARLES E. 315 NORTH HILLSIDE.67214
682-3221
37 M 1902 63 ORS
HENSLEY JR, CLINE D. 3244 E DOUGLAS.67208
689-9272
20 M 1902 44 ORS
HERED, JOHN. 959 N EMPORIA.
262-3613
41 M 2802 67 N
HERSHORN, SIMON E. 3333 E CENTRAL.67208
68S-1291
22 M 1902 46 R
HIEBERT, ABRAHAM E. 1530 W 13.67203
-
94 M 2802 25 OO
HINSHAW, ALFRED H. 6110 ONEIDA.67208
-
07 M 1902 33 OO
HINSHAW, CHARLES T. 256 N BLECKLEY DR.67208
682-8651
99 M 4705 26 OO
HIRATZKA, TOMIHARU. 3900 N WOODLAWN.67214
685-2151
13 M 511 43 PATH
HODGSON, ROBERT B. 841 NORTH BROADWAY.67207
263-6131
22 M 1902 47 FP
HODSON, HERVEY R. 1122 S CLIFTON.67218
682-S012
03 M 1606 31 GS
HOLDEN JR, RAYMOND F. 262 SOUTH BROOKSIDE.67208
683-1932
10 M 2802 33 OO
HOLLADAY, HARMON M. 337 N RUTAN.67208
681-0431
23 M 1902 S2 ANES
HORBELT, DOUGLAS V. S50 NORTH HILLSIDE.67214
682-6511
47 M 4802 72 OBG
* HOUSHOLDER, DANIEL F, 925 N ST FRANCIS, 67214
262-6211
43 M 1902 70 R-NM
HOUSHOLDER, MARTHA S. 835 NORTH HILLSIDE.67207
-
46 F 1902 72 D
HOWARD, DONALD O. 959 N EMPORIA.67214
265-7241
11 M 1902 38 OPH
HUEBERT, DEAN A. 5025 E KELLOGG.67218
682-1534
22 M 1902 46 FP
HULL, KENNETH L. 1035 N EMPORIA.67214
265-7903
38 M 2301 69 P
HULTGREN, MYRON K. 855 N HILLSIDE.67214
685-1381
41 M 1902 68 FP
HUME, JOSEPH W. 3243 E MURDOCK.67208
685-2223
38 M 1902 69 OBG
HUMMER, LLOYD M. 3244 E DOUGLAS.67208
689-9323
32 M 3901 57 IM
HURST, THOMAS C. 959 N EMPORIA.67214
264-3543
14 M 1902 38 PD
HUSTEAD, ROBERT F. 327 N RUTAN.67208
681-0431
28 M 801 S4 ANES
HYNDMAN, H HAROLD. 7015 ROCKWOOD RD.67206
-
10 M 1902 36 OO
HYNES, HENRY E. 410 N ROOSEVELT.67208
262-4467
35 M 53902 58 HEM
IBARRA, J LUIS. 1035 N EMPORIA.67214
262-1853
20 M 64901 46 P
JACKSON, CHARLES R. 1035 N EMPORIA.67214
263-0812
27 M 1606 53 GS
JAMES, VERNON L. 3333 E CENTRAL, 67208
268-8302
29 M 3601 55 PO
JAWADI, M HUSAIN, V A 5500 E KELLOGG.67218
685-2221
47 M 49521 71 IM
JAZAYERLI, NABIL. 5105 EAST 21ST.67208
681-2401
44 M 87501 70 CD

JEHAN, SAYED S, MENTAL HEALTH CLINIC,67214
268-8251
33 M 70403 59 P
JENNEY, CHARLES B, 905 N EMPORIA,67214
263-2795
34 M 2834 61 GS
JESTER, SHELBY L, 3243 EAST MURDOCK #101,67208
-

JOHNSON, THOMAS E, 3333 E CENTRAL,67208
685-1291
41 M 1643 67 R
JORDON, DAVID W, 3243 EAST MURDOCK #404,67208
682-6585
45 M 1611 71 1M
JUDILLA, FRANCISCO JR, 2322 EAST CENTRAL,67214
263-6186
44 M 74801 71 A
JUNOD, FORREST LEE, 3244 E DOUGLAS,67208
689-9124
M 1902 65 TS
KADISON, HERBERT I, ST FRANCIS HOSP,67214
262-6211
44 M 1611 69 R
KAELSON JR, PAUL A, 2012 W 13TH,67203
943-8671
17 M 1902 50 FP
KAROATZKE, E STANLEY, 1301 N WEST ST,67203
943-9342
39 M 1720 64 FP
KARDATZKE, JON K, 1301 N WEST ST,67203
943-3271
36 M 1720 62 FP
KASHA, ROBERT L, 1442 N OLIVER,67208
684-0181
11 M 2834 38 S
KASSEBAUM, KENNETH G, 3420 EAST DOUGLAS,67208
685-6381
1606 60 CHP
KAUFMAN, EUGENE E, 3243 E MURDOCK,67208
685-1491
30 M 1902 56 ORS
KEENE, GEORGE H, 502S E KELLOGG,67218
682-1534
20 M 1902 49 GS
KEENY, M GARY, 841 N BROADWAY,67214
263-6131
36 M 1902 66 FP
KELLER, JAMES P, 1431 SOUTH BLUFFVIEW,67218
685-1284
48 M 1902 74 1M
KELLY, ROBERT W, 3333 EAST CENTRAL #301,67206
682-6511
46 M 4802 72 OBG
KENDALL, TOM E, 825 N EMPORIA,67214
262-3495
37 M 3901 62 PS
KENDRICK, J GILLERAN, 3333 E CENTRAL,67208
682-6511
20 M 1902 46 OBG
KENNEDY, GERALD T, 2916 E CENTRAL,67214
684-5243
35 M 1902 61 GE
KHICHA, GYANCHAND J, 905 N EMPORIA,67214
263-0296
37 M 49S30 61 TCDS
KHOURY, GEORGE H, 3333 E CENTRAL,67208
681-2021
32 M 33002 55 PD
KIM, PAIK N, 1035 N EMPORIA,67214
262-4467
33 M 58302 58 HEM
KIM, SOON EUNG, 114 E WILSHIRE TERR,67208
686-2193
27 M 58301 48 PATH
KIMBLE, JAMES A, 3244 E DOUGLAS, 67208
689-9316
45 M 702 71 OPH
KIRK JR, E DAVID, 1431 S BLUFFVIEW DRIVE,67218
685-13S1
34 M 1902 62 1M
KISER, JOHN L, 3243 E MURDOCK,67208
685-6222
37 M 2802 62 GS
KISER, WILLARD J, 3243 E MURDOCK,67208
685-6222
05 M 4705 30 GS
KITCHEN, ROBERT R, 3420 E DOUGLAS,67208
685-2355
26 M 1902 52 CHP

KLAUS, HANNA, ST FRANCIS HOSP,67203
262-6211
28 F 2002 50 OBG
KNAPP, LESLIE E, 302 SOUTH CRESTWAY,67218
-
96 M 1902 25 OO
KNAPP, M ROBERT, 1128 SOUTH CLIFTON,67211
684-7251
23 M 3519 47 ANES
KNAPPENBERGER, ROY C, 3244 E DOUGLAS,67208
689-9327
16 M 1902 41 PD
KNEIDEL, THOMAS W, 925 N EMPORIA,67214
262-3476
40 M 4101 66 ORS
KOURI, SAMMY H, 3243 E MURDOCK,67208
682-2911
33 M 3901 57 GS
KRAUSE, ROLAND L, 855 N HILLSIDE,67214
685-1381
25 M 1902 53 IM
KURTH, C JOSEPH, 925 N EMPORIA,67214
263-6177
10 M 3006 35 NP
KUTILEK, FRANK J, 10300 WEST CENTRAL,67212
722-4258
30 M 1902 57 FP
LAHAM, ALEXANDER J, 2723 BLVD PLAZA,67211
683-4658
20 M 1902 44 1M
LAI, JENG Y, 111 N ST FRANCIS,67214
265-2613
41 M 38502 67 TS
LANCE JR, JOHN F, 3244 E DOUGLAS,67208
689-9438
20 M 1902 45 ORS
LATIMER, KATHERINE, 3243 EAST MURDOCK,67206
686-7327
49 F 1205 75 ANES
LAUVER, MARY ANN, 1001 NORTH MINNEAPOLIS,67214
268-8302
40 F 1902 74 PD
LAWN, RAYMOND A, 715 N MISSION RD,67206
683-8991
09 M 2604 35 AM
LAZAR, HARRY, 511 N HILLSIDE,67214
684-0501
08 M 1611 35 A
LEE, R REX, 6155 E HARRY,67218
685-2306
29 M 3901 55 FP
LEE JR, EDWARD S, 2002 E 17TH ST,67214
264-8273
09 M 4707 37 FP
LEIKER, JOSEPH, 550 NORTH HILLSIDE,67214
-
1M
LEISY, JERALD W, P O BOX 8037,67208
684-0201
42 M 1902 68 P
LIES, JAMES E, 3244 E DOUGLAS,67208
689-9240
43 M 1902 69 IM
LIN, JOE J, 929 N ST FRANCIS,67214
262-6211
42 M 24404 69 PATH
LINDLEY, MILTON E, 657 S TERRACE DR,67218
-
14 M 1902 50 OO
LINHARDT, RONALD D, 3243 E MURDOCK,67208
683-2655
36 M 2803 64 OBG
LITTLE, L GILBERT, 122 S WESTFIELD AVE, 67209

99 M 401 27 OO
LOCKHART, JOSEPH G, 5900 E CENTRAL,67208
684-7239
17 M 4113 43 PD
LOEFFLER, JAMES A, 400 N WOODLAWN,67206
685-5375
36 M 3841 63 A
LOEWEN, HENRY H, 2142 WEST 17TH,67203
-
03 M 1902 35 OO
LOEWEN, WILLIAM C, 1301 N WEST ST,67203
943-3253
41 M 1902 71 FP
LOGAN, GEOFFREY G, 345 N HILLSIDE,67214
682-4572
31 M 14303 56 OBG

LORCH, VICHIEN, 1001 N MINNEAPOLIS, 67214
268-8302
40 M 89101 65 PO
LOVETT, PAUL A, 110 PATTON,67214
683-6683
09 M 1902 45 ORS
LOW, HAROLD L, 1148 S HILLSIDE,67211
684-2858
18 M 1902 44 FP
LUEKEN, LUEKE B, 3244 E DOUGLAS,67208
689-9234
23 M 40723 52 OBG
LUELLEN, THOMAS J, 3244 E DOUGLAS,67208
689-9244
17 M 1902 41 IM
LUZZATI, ENZO F, ST FRANCIS HOSPITAL,67214
262-6211
25 M 56119 50 R
MADISON JR, WARD N, 3400 E GRAND,67218
686-9432
37 M 3601 62 PATH
MAGIDSON, ELLIOT ARTHUR, 424 N WOODLAWN,67208
686-2193
43 M 1611 68 PATH
MANNING, ROBERT T, 2221 NORTH HILLSIDE,67219
689-3193
27 M 1902 54 IM
MANSOUR, BADIE S, 3243 E MURDOCK S 101,67208
686-7327
45 M 33002 69 A
MARSH, HENRY O, 925 N EMPORIA,67214
262-3476
18 M 1611 43 ORS
MARTIN JR, GLEN E, 2322 E CENTRAL,67214
263-6186
20 M 1902 49 ANES
MARYMONT JR, JESSE H, WESLEY MED CENTER,67214
685-2151
28 M 3515 54 PATH
MASTIO JR, GEORGE J, 3243 E MURDOCK,67208
684-5235
25 M 1902 52 GS
MATASSARIN, BENJAMIN M, 2916 E CENTRAL,67214
684-5243
20 M 1902 45 IM
MATASSARIN, FREDERICK W, 734 N EMPORIA,67214
265-2382
15 M 1902 37 U
MAWDSLEY, MICHAEL W, 3333 EAST CENTRAL #610,67208
686-6659
49 M 1902 74 PD
MCCLANAHAN, WARD A, 5105 E 21ST,67208
684-8211
22 M 3005 48 ORS
MCCLELLAN, ERNEST L, 3243 E MURDOCK,67208
686-7327
38 M 4802 70 ANES
MCCOY, CHARLES P, 3333 E CENTRAL,67208
682-6511
17 M 3006 42 OBG
MCGUIRE, WILLIAM F, 3333 E CENTRAL,67208
683-5655
17 M 4101 43 PD
MCMAHON, MERRI M, 1001 NORTH MINNEAPOLIS,67214
248-8378
46 F 5404 72 IM
MEADOWS, DONALD C, 3333 E CENTRAL 504,27208
683-5611
36 M 102 69 OPH
MEEKER II, BRUCE P, 345 N HILLSIDE,67214
686-3384
30 M 1902 58 OBG
MEHRA, PROMILLA, 2810 EAST 21ST STREET,67214
-
40 F 63 GP
MELEAN, JAIME, 1152 SOUTH CLIFTON,67208
681-2401
40 M 65 CD
MELVIN, DAVID B, 905 NORTH EMPORIA,67214
263-0296
42 M 4706 67 TS
MENAKER, JEROME S, 2703 E CENTRAL,67214
685-1227
16 M 1005 41 OBG
MENDIONES, L MARLENE, 835 N HILLSIDE,67214
685-4395
45 F 1611 70 D
MENDIONES, RUPERTO D, 3243 E MURDOCK ST 404,67214
-
M 1M

MENEHAN, H JAMES, 3244 E DOUGLAS,67208
689-9404
26 M 1902 53 PD
MENKING, F W MANFRED, 3244 E DOUGLAS,67208
689-9336
34 M 40715 61 PD
MERCADER, MARIO S, 2322 E CENTRAL,67214
263-6186
43 M 74801 64 A
MEREDITH, W TOM, 1035 N EMPORIA,67214
263-7285
35 M 4812 61 IM
MERRITT, JOE P, 2703 E CENTRAL,67214
685-1277
46 M 2802 71 OBG
MERSHON, JAMES C, 1035 N EMPORIA,67214
263-3271
37 M 1803 63 IM
MEULBROEK, HARVEY J, 3244 E DOUGLAS,67208
689-9156
31 M 5605 56 A
MEYER, WARREN E, 1144 S CLIFTON,67218
684-5237
27 M 1606 51 S
MICHELBACH, ALBERT P, 2916 E CENTRAL,67214
684-5243
35 M 2101 61 IM
MILLER, DON E, 4145 E KELLOGG,67218
682-6551
20 M 2802 44 S
MILLER, LAWRENCE H, 1131 SOUTH CLIFTON,67218
685-4354
40 1001 67 FP
MILLER, PHILIP A, 51-5 EAST 21ST,67208
684-2081
44 1902 70 GS
MILLS, CHARLES D, 1144 S WATER,67213
-
89 M 2002 14 OO
MIRZA, MEDO, 3333 E CENTRAL,67208
686-6683
38 M 40733 64 PDS
MITCHELL, ALEX C, PO BOX 42,67201
267-6276
18 M 1902 50 FP
MOORE, DENNIS F, 1035 N EMPORIA,67214
265-3226
36 M 2101 62 HEM
MORGAN, DICK A, 3243 E MURDOCK,67208
686-7327
43 M 3901 69 ANES
MORGAN, JAMES I, 3124 S SENECA,67217
522-2266
29 M 1606 53 FP
MORGAN III, LOUIS S, 8030 E KELLOGG,67207
683-3811
22 M 3901 48 FP
MORRISON, RICHARD L, 1148 S HILLSIDE,67211
683-6506
42 M 1902 67 FP
MORROW, THOMAS F, 3310 E DOUGALS,67208
685-1443
21 M 5606 46 P
MOSELEY, JACK E, 1120 S CLIFTON,67218
682-4982
25 M 3901 53 FP
MOSIER, STANLEY JAY, 3243 E MURDOCK,67208
685-8231
42 M 1902 68 FP
MOTEKALLEN, MOHAMMED, 1111 NORTH ST FRANCIS,67206
265-2613
44 M 51701 68 IM
MOY, JAMES T, 3028 E 2ND,67214
682-1392
14 M 4102 40 1M
MUELLER, VERNETTE A, 1431 S BLUFFVIEW,67218
684-3981
17 M 2802 41 OBG
MULLINIX, JANICE M, 3243 MURDOCK,67208
685-2377
47 F 3006 73 N
MURPHY, BARRY L, 3243 EAST MURDOCK #500,67208
684-0251
45 M 1902 71 1M
MURPHY, DUANE A, 3243 E MURDOCK,67208
685-1491
32 M 1902 65 ORS
MURPHY, PAUL M, 3600 E HARRY,67208
685-1111
28 M 3006 51 R

MURRAY, KENT 8, 841 NORTH BROADWAY,67206
263-6131
47 M 3901 73 IM
NEGRON, ANGEL G, 3244 EAST DOUGLAS,67208
689-9369
45 4201 70 HEMO
NELSON, GERALD O, 825 N EMPORIA,67214
262-3495
34 M I902 60 PS
NELSON, RUSSELL ALAN, 3333 E CENTRAL,67208
685-5271
18 M I902 45 PD
NELSON JR, GUST H, 3400 GRAND AVE,67218
685-1111
23 M 1902 46 OR
NESMITH, LESLIE W, 3333 E CENTRAL,67208
683-5611
40 M I902 66 OPH
NEWCOMB, WARD M, I300 E 13TH,67214
263-0296
34 M 1902 59 TS
NEWSOM, F CARTER, 3310 E DOUGLAS,67208
685-1443
18 M I201 43 P
NIXON, WILLIAM A, 3333 E CENTRAL,67208
683-6622
16 M I902 44 S
NORRIS, REBECCA M, 3214 E DOUGLAS,67208
689-9111
42 F I611 70 IM
NORRIS, ROBERT P, 3244 E DOUGLAS,67208
689-9232
17 M I902 43 IM
NORTH, DORIS G, 1148 S HILLSIDE,67211
684-5257
16 F I902 47 FP
NORTH, VICTOR, 1148 S HILLSIDE,67211
684-5257
18 M I902 47 FP
NORTON, ROBERT K, 3244 E DOUGLAS,67208
689-9235
32 M I001 57 PD
NOWLIN, NANCY S, I001 N MINNEAPOLIS,67214
268-8378
F I902 74 IM
NYBERG, FREDRIK F, 3333 E CENTRAL,67208
682-6511
22 M 2101 46 GYN
O DONNELL JR, LEONARD A, 8033 E DOUGLAS,67207
684-2835
27 M I902 55 IM
O DONNELL SR, LEONARD A, 8033 E DOUGLAS,67207

02 M 5606 26 OD
OCHSNER, BRUCE 8, I035 N EMPORIA,67214
263-6273
39 M I902 65 OPH
ODENHEIMER, BURTRAM J, 3244 EAST DOUGLAS,67208
689-9137
48 M 2105 73 N
OSBORNE, CONRAD C, 855 N HILLSIDE,67214
685-1381
38 M 1902 67 FP
OSIO, ANTONIO L, I520 S CLIFTON,67206
685-2371
41 M 26404 65 FM
OSUNA, WILLIAM G, 2525 W 13TH,67203
943-9391
25 M 2802 51 FP
OWEN, LARUE W, 303 S HILLSIDE,67211
684-7251
19 M 1902 50 ANES
OWEN, PERRY A, 303 S HILLSIDE,67211
684-7251
37 M I902 64 ANES
PADGETT, WILLIAM L, 841 S HILLSIDE,67211
683-7503
24 M 1902 51 FP
PAGE, RUTH G, 1148 S HILLSIDE,67211
684-0255
13 F 1902 43 IM
PALMER, DAVID L, P O BOX 2858,67201
686-7161
37 M I902 63 PATH
PARKER, HAROLD L, 2932 BENJAMIN, 67204
262-3765
32 M 1902 67 FP
PARMLEY, CHARLES C, 5025 E KELLOGG,67218
682-1534
18 M 1902 44 S

PATTERSON, BRUCE W, 1520 SOUTH CLIFTON,67218
685-2371
46 M I902 73 EM
* PAY, NORMAN T, 929 N ST FRANCIS, 67214
262-6311
45 M 74802 68 N
PENCE, CHARLES O, 3244 EAST DOUGLAS,67208
689-9195
42 M 1902 68 ORS
PENNINGTON, KATHERINE, 2113 SO BLUFF CT,67218
685-5271
16 F I902 43 PD
PETERIE, JERRY P, 1001 N MINNEAPOLIS,67214
268-8378
48 M I902 75 IM
PETERS, DALE W, 3201 EAST SECOND,67208
682-9241
21 M I902 45 PSY
PETTERSON, D RUTH S, 5025 EAST 21ST,67208

19 F I902 44 OD
PHIPPS, JACK G, 315 N HILLSIDE,67214
686-3391
21 M I902 53 FP
PINSKER, JACOB A, I035 N EMPORIA,67214

06 M 1902 35 OD
POOREBARAC, LEONARD F, I035 N EMPORIA,67214
263-3143
24 M 3006 53 FP
POLING, TERRY L, 615S E HARRY,67218
685-2306
36 M I902 62 FP
POLLACK, SIMON, 7077 EAST CENTRAL,67206

M 36 OD
POLLOCK, ANTHONY G A, I035 NORTH EMPORIA #290,67214
265-1631
45 M 8305 71 OS
POOLE, BERNARD T, I035 N EMPORIA,67214
264-2806
37 M 53902 62 ORS
PORTER, GARRY L, 3242 E MURDOCK, 67208
686-7351
35 M I606 61 PSY
POWERS, K DEAN, 2703 E CENTRAL,67214
685-1277
23 M I902 47 OBG
PRIETO, LUIS E, 959 N EMPORIA SUITE 403,67214
265-2613
39 M 26407 65 IM
* PRUITT, JOHN C, 427 N HILLSIDE, 67214
686-6608
48 M 1902 74 OTO
PULLMAN, NORMAN K, 3007 E CENTRAL,67214
686-7369
21 M 3006 45 PS
PURINTON, LEW W, 345 N HILLSIDE,67218
685-1301
23 M 1902 48 IM
PURVES, ROBERT K, 37 MISSION,67206
685-2371
18 M I606 43 S
PUTNAM, LYLE 8, 959 N EMPORIA,67214
262-5037
11 M I902 36 FP
RAHBAR, AL A, 905 NORTH EMPORIA,67214
263-0296
43 M 2501 69 TS
RANDALL, GEORGE R, 5025 EAST 21ST,67208
684-2838
43 M 2802 69 OTO
RANDLES, MICHAEL J, 959 N EMPORIA,67214
265-0789
48 M I902 73 IM
RAUSA JR, FRANCISCO C, 1148 SOUTH HILLSIDE,67211
683-4658
42 M 74808 66 IM
RAWCLIFFE JR, ROBERT A, 925 N EMPORIA,67214
262-3476
29 M 3501 55 ORS
RAZEK, ZACK A, 905 NORTH EMPORIA,67214
263-0296
46 M 60501 70 CDTS
REALS, WILLIAM J, ST JOSEPH MED CENTER,67218
686-9432
20 O 3006 45 PATH
REAZIN, WALTER L, 1430 HOMESTEAD,67208
685-1381
30 M 1902 58 FP

REED, A J, 1520 S CLIFTON,67218
685-2371
40 M 3901 65 EM
REED, O CRAMER, KUMS,67214
268-8202
15 M 2802 41 ADM
REED, DAVID D, 3333 E CENTRAL,67208
685-1291
43 M 1902 69 DR
REEVES, BRADFORD F, 3333 E CENTRAL,67208
685-1291
37 M 4812 62 R
RELIHAN, DONALD A, 3333 E CENTRAL,67208
684-5158
27 M 1902 54 DPH
REMPEL, JOHN H, 3333 E CENTRAL,67208
685-1812
38 M 3901 62 PS
RENNEBOHM, ROBERT M, 1001 NORTH MINNEAPOLIS,67214
268-8302
46 M 502 72 PD
RHOADS, JAMES P, 420 NORTH MISSION,67206
689-9325
34 M 3520 60 IM
RHODEN, CURTIS H, 3243 E MURDOCK,67208
684-0252
33 M 1803 59 IM
RHODES, IVAN E, 3333 E CENTRAL,67208
685-1291
25 M 3901 49 R
RHODES, LOWELL M, 315 N HILLSIDE,67214
685-1461
25 M 1902 53 FP
RICHARDSON, STEWART F, 3420 E DOUGLAS,67208
685-2321
28 M 3005 54 P
RIEGER, ERNEST H, 3243 E MURDOCK,67208
682-4591
29 M 1902 56 GS
RINDT, PHILLIP, ST FRANCIS HOSP,67214
262-6211
M 1902 71 FP
RIORDAN, HUGH D, 434 N OLIVER,67208
682-9241
32 M 5605 57 P
ROBERTS, DANIEL K, WESLEY MED CENTER,67214
682-6511
36 M 3005 61 OBG
ROBERTS, LOUIS S, 115 S RUTAN,67218
-
95 M 2834 20 DD
ROBERTSON, JOSEPH K, 905 N EMPORIA,67214
263-0296
41 M 3901 66 GS
ROBINSON, GEORALD, 3333 E CENTRAL,67208
686-6659
28 M 1902 54 PD
ROBINSON, JOHN E, 3243 E MURDOCK, 67208
686-7351
32 M 6201 50 P
ROBINSON, ROBERT H, 3244 E DOUGLAS,67208
689-9445
20 M 1902 53 ANES
ROGL, DAVID A, 925 N EMPORIA,67214
262-3765
48 M 1902 74 FP
RODRIGUEZTOCKER, LILIA, 1111 N ST FRANCIS,67214
265-2613
21 F 27501 49 IM
ROMALIS, BRIAN E, 3420 E DOUGLAS,67208
682-5069
39 M 6201 63 P
ROSE, SHELBY D, 3333 E CENTRAL,67208
681-2741
40 M 2012 68 PATH
* ROSEN, DAVID, 1035 N EMPORIA, 67207
265-3226
48 M 1902 74 PD
ROSENBERG, THOMAS F, 611 N HILLSIDE,67214
684-0501
41 M 1642 68 A
ROSS, DENNIS LEE, 1035 N EMPORIA 105,67211
263-7285
47 M 5003 73 NEPH
RUSSELL, PHILIP W, 3244 E DOUGLAS,67208
689-9351
22 M 1902 44 IM
SABIN JR, GEORGE M, 321 N TOPEKA,67202
265-6601
12 M 5002 39 ADM

SADIQ, SULEMAN, 1111 N ST FRANCIS,67214
265-2613
40 M 70401 63 TS
SAEED, MOHAMMAD A, 1125 S CLIFTON,67218
685-2371
42 M 70404 66 IM
SALGADO, CARLOS R, 111 NORTH ST FRANCIS,67214
265-2613
33 M 13201 55 CD
SANTOSCOY, GILBERT S, 3244 E DOUGLAS,67208
689-9124
38 M 4812 62 S
SCANLAN, TIMOTHY M, 1122 N TOPEKA,67208
265-2876
46 M 2604 71 FP
SCHILTZ, FRANCES, 115 S RUTAN #108,67218
-
93 F 6701 23 DD
SCHLACHTER, ERNEST R, 406 E CENTRAL,67202
265-0705
24 M 1902 52 FP
SCHLICHER, JOHN E, 3244 E DOUGLAS,67208
689-9346
40 M 1803 66 O
SCHLUETER, JOHN J, 3333 E CENTRAL,67208
685-9289
31 M 3841 56 R
SCHNELLE, JOACHIM, 4145 EAST KELLOGG,67218
682-0621
44 40933 70 FP
SCHOPF, CLIFTON C, 425 E MURDOCK, 67214
262-3765
29 M 1902 57 FP
SCHULZ, MILLARD E, 1155 EMERSON,67212
263-6131
12 M 1902 45 DM
SCHWARTZ, V DEAN, 400 N WOODLAWN,67208
684-3881
24 M 1902 48 FP
SCOTT, VINCENT L, 72 MISSION RD,67207
-
03 M 3806 29 DD
SCOTT, WILLIAM H, 1431 S BLUFFVIEW, 67218
685-1111
41 M 4901 65 CD
SHAFER, PRESTON J, 3333 E CENTRAL,67208
682-6511
20 M 3005 46 OBG
SHAH, MUKHTAR H, 1801 EAST 10TH STREET,67207
268-8262
40 70404 63 P
SHAW, JAMES W, 150 S BATTIN,67218
-
98 M 1601 27 DD
SHAW, RICHARD C, 825 N EMPORIA,67214
262-3495
35 M 1902 61 PS
SHELLITO, JOHN G, 3244 E DOUGLAS,67208
689-9124
18 M 1606 43 TS
SHIFLET, ALBERT W, 841 N BROADWAY,67214
-
06 M 3901 33 DD
SHRADER, ODYLE A, 3333 E CENTRAL,67208
682-4851
16 M 1902 41 EENT
SIEGEL, ALBERT R, 3600 E HARRY,67218
685-1111
22 M 1642 47 PM
SIFFORD, R LAWRENCE, 959 N EMPORIA,67214
265-0561
25 M 1803 52 IM
SKIBA, RICHARD M, 3244 E DOUGLAS,67208
689-9477
43 M 5606 70 GE
SLEEPER, CAROL A, 3244 EAST DOUGLAS,67208
689-9275
33 F 1902 58 PATH
SLEEPER, DONALD C, 929 NORTH ST FRANCIS,67214
265-1684
32 M 1902 58 R
SMITH, ALVIN L, 929 N ST FRANCIS,67214
262-6211
28 M 5606 57 PATH
SMITH, ERMA A, 120 N KANSAS AVE,67214
-
95 F 1601 33 DD
SMITH JR, WILLARD J, 851 N HILLSIDE,67214
685-1371
32 M 1611 57 U

SNYDER, GREGG M, 3243 E MURDOCK,67208
685-2377
27 M 1803 S4 NS
SOLOMON, HERMAN, 835 N HILLSIDE,67214
685-4395
37 M 2701 62 O
SOLTZ, ROBERT A, 3244 EAST DOUGLAS,67208
689-9381
47 M 2803 74 PD
SOMERS, MARVIN M, ST FRANCIS HOSP,67214
262-6211
23 M 1902 48 R
SPANN, RICHARD W, 3243 E MURDOCK,67208
684-0252
40 M 1902 6S PUD
STANLEY, KENNETH E, 959 N EMPORIA,67214
267-0256
31 M 1902 56 U
STARK, JAMES R, 3244 E DOUGLAS,67208
689-9422
20 M 1902 44 R
STEIN, PAUL S, 3243 E MURDOCK,67208
685-2377
40 M 330S 66 NS
STEWART, JACK T, 481S E CENTRAL,67208
683-8401
30 M 2802 57 IM
STOLZ, ELMER G, 425 E MURDOCK,67214
265-6287
17 M 3006 4S FP
STOSKOPF, LAWRENCE E, 3333 E CENTRAL,67208
685-4389
39 M 1902 72 ANES
STRANGE, MICHAEL, 550 NORTH HILLSIDE,67214
-

STREET, DAVID E, 905 N EMPORIA,67214
263-0296
3S M 2101 6I GS
STRYKER, TERRY M, 3243 E MURDOCK, 67206
685-8231
47 F 1002 75 FP
SUERO, JESUS T, 1152 SOUTH CLIFTON,67218
681-1671
33 M 74802 PUD
SULLIVAN, CORNELIUS J P, 1431 BLUFFVIEW DR,67218
685-4851
18 M 3509 43 OBG
SULLIVAN, LEONARD L, 3244 E DOUGLAS,67208
689-9454
3S M 1902 61 PD
SWEENEY, JAMES G, 929 NORTH ST FRANCIS,67214
-

SWISHER, WILLIAM C, 5025 E KELLOGG,67218
682-1534
21 M 1902 47 OBG
TATAATI, DANIEL A, 1111 NORTH ST FRANCIS,67218
265-2613
44 M 49535 67 TS
TAYLOR, RICHARD J, 929 N ST FRANCIS,67214
262-1952
21 M 3006 49 PATH
TERRY JR, DANIEL W, 3333 EAST CENTRAL #214,67208
685-1291
4S M 502 71 R
THELEN, J CHRISTINE, 212 BITTING BLDG,67202
267-3936
13 F 5104 37 OBG
THOMPSON, DANIEL M, 118 E 21ST,67214
838-3381
19 M 1902 50 FP
TIHEN, EDWARD N, 3244 E DOUGLAS,67208
689-9481
24 M 1606 48 IM
TIHEN, HENRY N, 1227 N RIVER BLVD,67203
-
96 M 1601 19 OD
TILLER, GEORGE R, 5101 E KELLOGG,67218
684-5255
41 M 1902 67 AM
TILTON, FRANK M, 3244 E DOUGLAS,67208
689-9137
33 M 2002 59 N
TINKER, ROBERT C, 1126 S CLIFTON,67218
682-7578
31 M 2834 57 ORS
TINTEROW, MAURICE M, 3333 E CENTRAL,67208
685-4389
17 M 4802 41 ANES

TIPPIN JR, ERNEST E, 959 N EMPORIA,67214
265-5256
24 M 1902 50 OTO
TOCKER, ALFRED M, 1111 N ST FRANICS,67214
265-2613
1S M 4802 40 TS
TONN, GERHART R, 855 N HILLSIDE,67214
685-1381
16 M 1902 44 FP
TRACY, TERRY A, 3333 E CENTRAL,67208
686-7301
3S M 2803 6I OBG
TREES, DONALD P, 1403 S HYDRAULIC,67211
-
14 M 4101 39 OD
TRETBAR, HARVEY A, 3243 E MURDOCK,67208
684-0251
2S M 1902 52 IM
TREWEEKE, MICHAEL W, 2916 EAST CENTRAL,67214
684-5234
46 M 1902 72 IM
UHLIG, PAUL J, 3244 E DOUGLAS,67208
689-9191
28 M 1902 57 PD
VAN DER VLIET, STEPHEN K, 3244 E DOUGLAS,67208
689-9316
42 M 1606 68 OPH
VIN ZANT, LARRY E, 1431 BLUFFVIEW,67218
682-6532
10 M 1902 40 GS
VINZANT, WHITNEY L, 1431 SOUTH BLUFFVIEW #109,67218
686-1991
4S M 1902 71 GS
VORHEES, VICTOR J, 3243 E MURDOCK,67208
685-8231
36 M 1902 68 FP
VOTH, DOUGLAS W, 2221 NORTH HILLSIDE,67219
268-8378
34 M 1902 59 IM
WADUD, ABDUL, 1801 EAST 10TH,67214
268-8251
3S M 70409 60 P
WAKIM, PAUL E, 1035 NORTH EMPORIA #290,67214
265-1631
4S 2878 ORS
WALKER, WILLIAM L, 3333 EAST CENTRAL #214,67208
685-2151
44 M 1902 70 R
WALLACE, ROBERT W, 303 SOUTH HILLSIDE,67211
684-7251
28 M 3006 53 ANES
WALSH, RICHARD A, 1001 N MINNEAPOLIS, 67214
268-8224
25 M 3519 48 ADM
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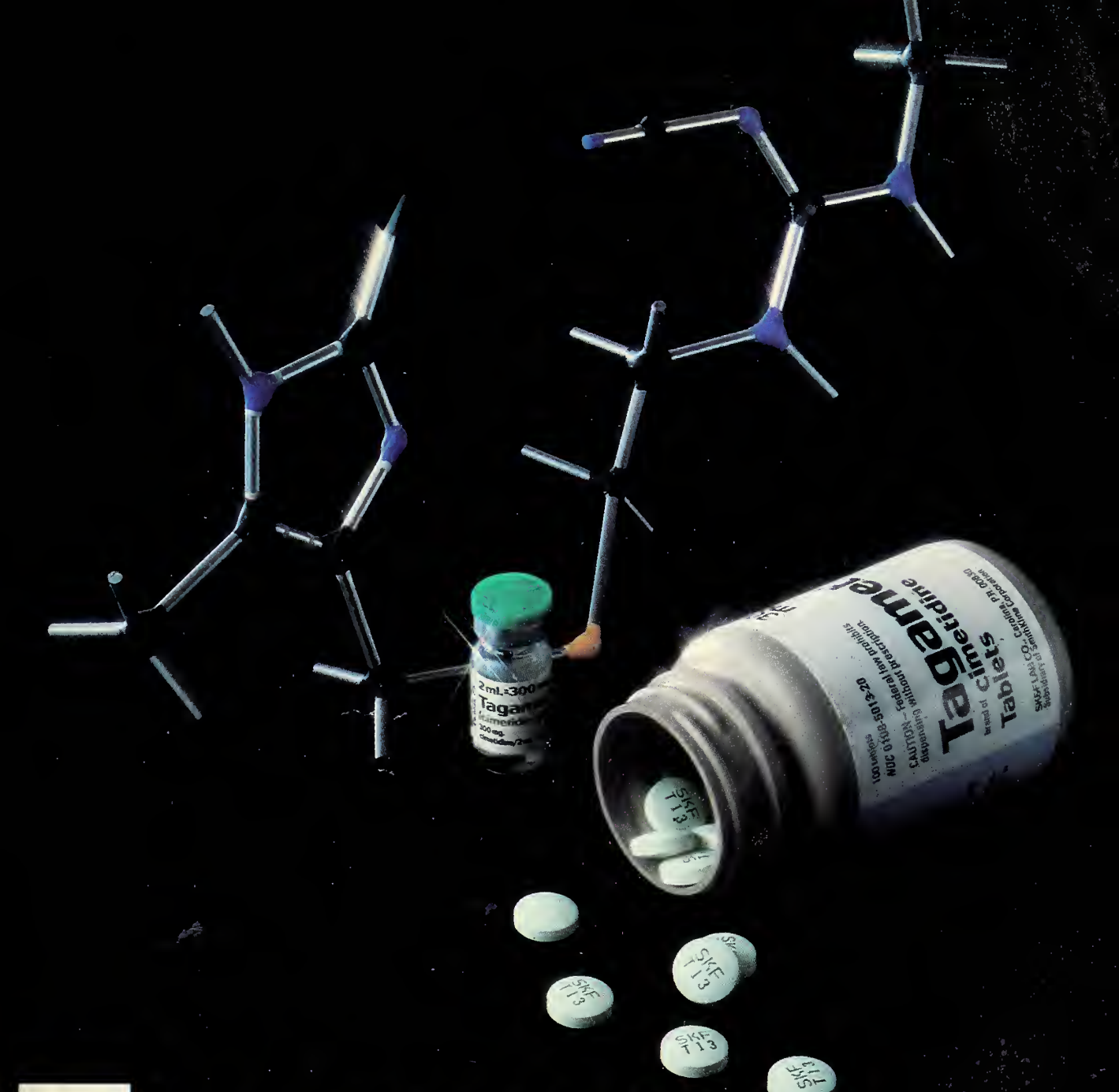
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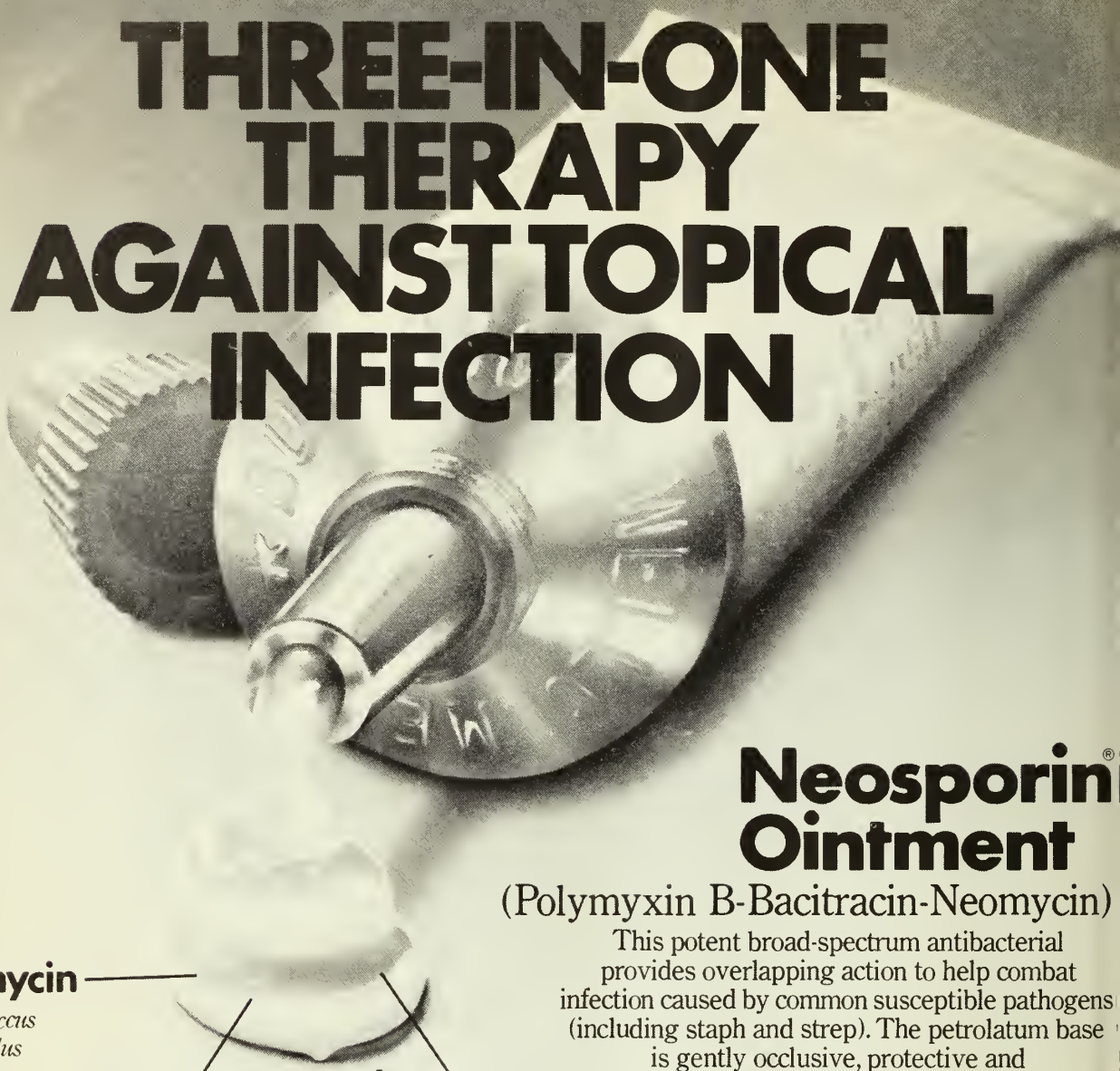
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WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

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Each tablet contains 160 mg trimethoprim and 800 mg sulfamethoxazole.

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Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* **It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination.** *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

The recommended quantitative disc susceptibility method (*Federal Register,* 37:20527-20529, 1972) may be used to estimate bacterial susceptibility to Bactrim. A laboratory report of "Susceptible to trimethoprim-sulfamethoxazole" indicates an infection likely to respond to Bactrim therapy. If infection is confined to the urine, "Intermediate susceptibility" also indicates a likely response. "Resistant" indicates that response is unlikely.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older:

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension,* containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

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Bactrim fights uropathogens in the urinary tract/vaginal tract/lower intestinal trac

Please see reverse side for summary of product information.

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

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Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

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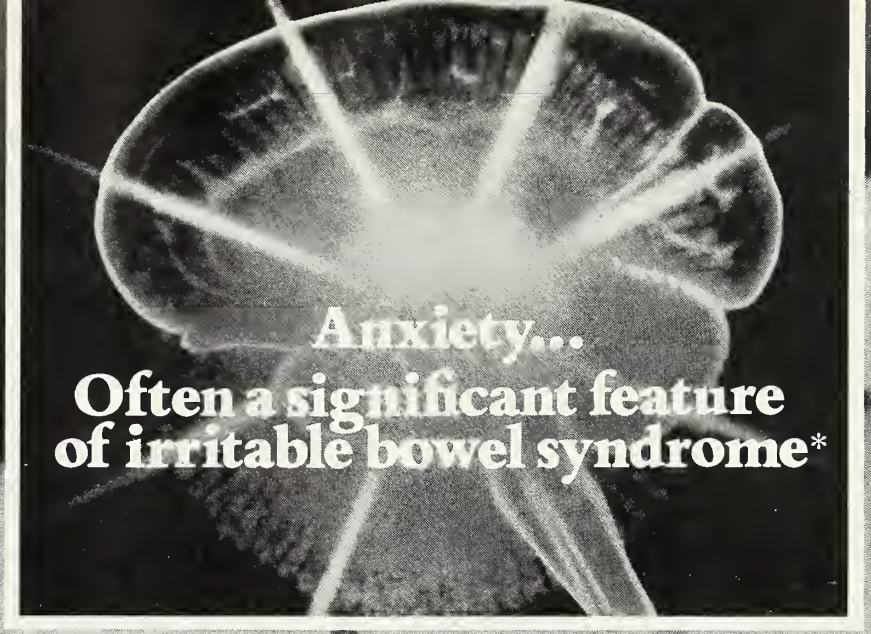
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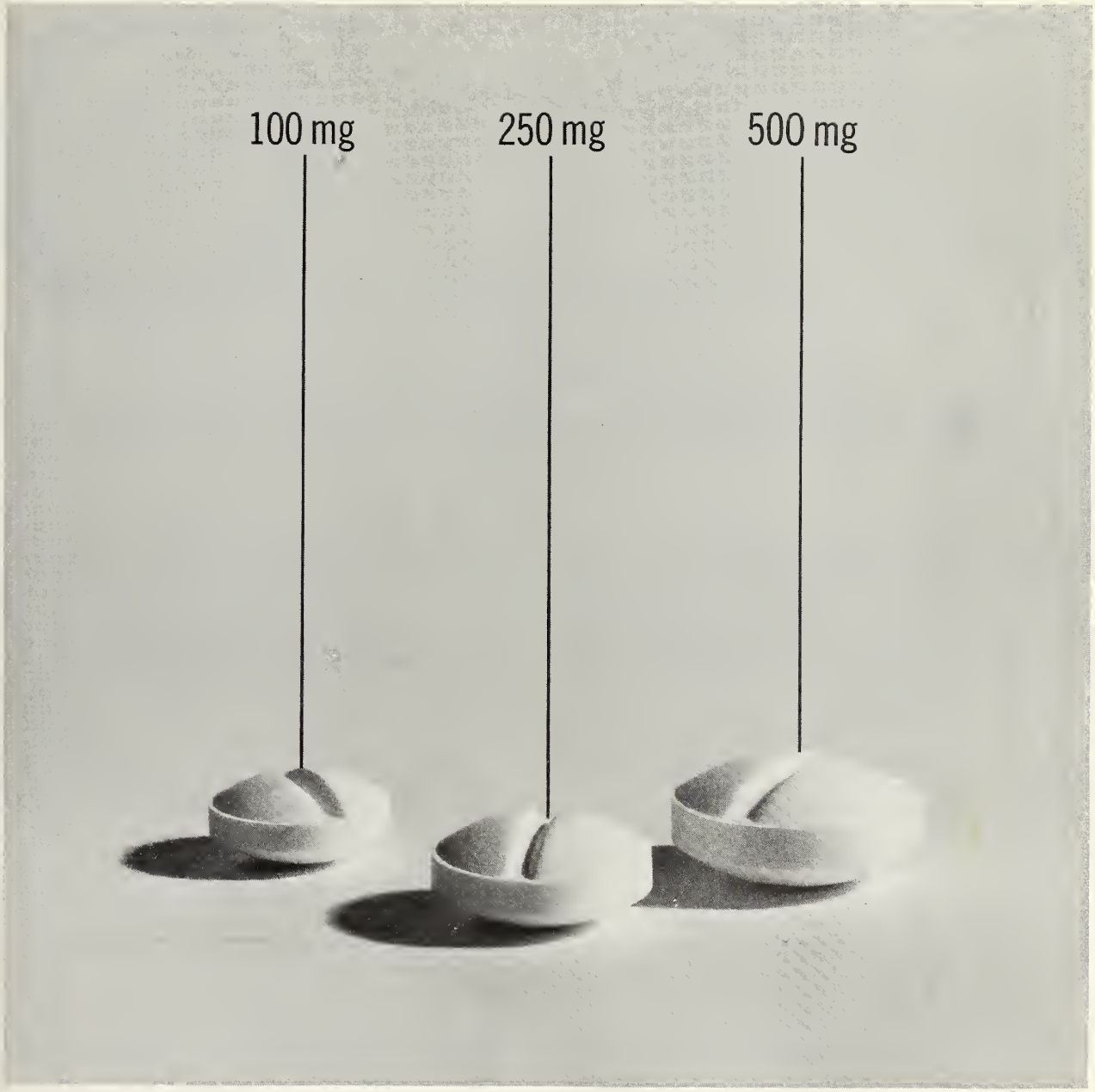
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Auxiliary News

June 18-21 saw approximately 40 Kansans in St. Louis for the AMA and AMAA annual meeting. A series of well planned workshops included legislation and information on hospital cost containment, national health insurance, membership — with some positive suggestions for increasing our numbers — health projects, medical marriages, and others.

Perhaps most stimulating of all the speakers was Keynoter Bjorn Secher, an authority on management and development. He presented thoughts on attitudes, power of positive minds, and communication in such a dynamic manner that one felt that any accomplishment was possible. His closing statements were: "Our goals become our potential worth. Nothing is impossible for the positive mind. Take the days as they come and make them the kinds of days you want." This is the philosophy that I hope will guide me and the Auxiliary through the coming year.

In July, the KMS Executive Committee approved unanimously the Auxiliary health education program, SCORE. Because this will be one of our emphasis programs, I would like to explain it briefly.

SCORE means "Student Council Offers Responsible Education." It was designed to involve students in peer education and can be implemented through student councils, FHA, Kayettes, or any other active youth organization. The material used is factual and accurate information, published by the National Foundation, March of Dimes, and deals with such subjects as drugs, alcohol, smoking, nutrition, venereal disease, virus infections, and the importance of immunization. The goal for this program is to provide good health education for our youth and to improve the quality of life for them and for their progeny. It is organized in such a way that the entire student body can be involved. The program, originated in Texas, has been used successfully in their schools for the past two years, and they will continue to use it. The National Student Council Association has approved the program. Although it was designed for senior high school students — that is where we will start — it can be adapted for the junior high school level. A workshop on SCORE was presented at our summer board meeting and will be given again during our statewide workshops in Manhattan (October 4) and Dodge City (October 5). We solicit your support of this program in your own communities, and welcome any suggestions you might have.

Sincerely,
Jean Crouch
President
Kansas Medical Society Auxiliary



Book REVIEWS

SIDE EFFECTS, by Melvin J. Silverstein, M.D., and Karen Sperling Silverstein, Doubleday & Company, Inc., New York, 1978. 264 pages. $7.95.

MASTECTOMY—few medical procedures can rival its scope of emotional impact. The subject has been ambitiously explored in this book by the Silversteins—cancer specialist Mel and writer-director Karen.

As a novel, it is on the plus side; the plot is tight, the characters well drawn, and the style facile. As educational essay, it adequately delineates the medical process of mastectomy. At its most complex level—that of emotional/psychological ramifications—it graphically portrays many of the conflicts inherent in the loss of the primary credential of womanhood.

A possible weakness is its attempt to answer some questions that it need not ask; after all, how many victims of breast cancer are involved in an overt relationship with a physician?

Also questionable is the characterization of the patient, who is imaged as young, physically beautiful, and with a voluptuous body. Although it can be theorized that loss of a breast might be exceptionally traumatic for such a woman, the fact remains that she is not typical.

However, these flaws are minor, and restructuring the book to eliminate them might diminish its impact as a novel.

Although different readers will extract different cogencies, *Side Effects* provides an effective medium for deeper insight into a contemporary concern.—*E.B.*

HIGH THERMAL STRESS

Although blood samples are still being analyzed, researchers at Kansas State University, Manhattan, have pinpointed the highest temperatures and humidity levels that a majority of people can tolerate.

The K-State researchers (Dr. Frederick H. Rohles, director of the KSU Institute for Environmental Research, where the studies were made; Dr. Stephan A. Konz, professor of industrial engineering; Dr. William B. Zuti, professor of health, physical education, and recreation; and Dr. Joseph Smith, professor of pathology in the College of Veterinary Medicine) found that being able to withstand high temperatures takes acclimation.

Younger subjects — men and women between the ages of 18 and 24 who had spent a great deal of time outdoors in the summer — were better able to tolerate higher temperatures.

Under the worst conditions observed (122F and 20% relative humidity, a condition similar to that in the desert) only one in four of these younger subjects was able to finish a four-hour experiment. Only one in eight of older subjects passed the four-hour test.

The next level tested (122F with 10% relative humidity) was not as taxing, but still not everyone could withstand the heat.

The toughest test that all participants could withstand was 80F and 50 per cent humidity, a condition found along US coasts in the summer.

The K-State experiment, called "Hematological Evaluation during High Thermal Stress," was financed by the US Air Force, and involved 262 participants who were tested on heat and humidity tolerance. They also were checked to see how various heat and humidity conditions affected their blood.

Participants ate a standard breakfast and were given a physical examination, which included measurement of skin and rectal temperatures, amount of oxygen used, and perceptual-motor coordinator prior to their four-hour ordeal in a heat-humidity controlled room. Men wore shorts; women wore shorts and halter straps. Rohles classified the study as "sedentary" and "semi-nude."

Rohles said subjects were taken from the room if their body temperature raised two degrees from what it had been at the beginning of the experiment.

The investigators feel that the study defines valid maximum tolerance levels and hope to investigate the relationship of other factors such as water replacement, protective clothing, exercise, acclimation, and the time of day.



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Possibly Effective:

1. For the relief of symptoms associated with cerebral vascular insufficiency.
2. In peripheral vascular disease of arteriosclerosis obliterans, thromboangiitis obliterans (Buerger's Disease) and Raynaud's disease.

Final classification of the less-than-effective indications requires further investigation.

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Intramuscular: 5 to 10 mg. (1 or 2 ml.) two or three times daily. Intramuscular administration may be used initially in severe or acute conditions.

Contraindications and Cautions: There are no known contraindications to oral use when administered in recommended doses. Should not be given immediately postpartum or in the presence of arterial bleeding.

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Precautions: Use with caution in patients with cardiac disease, hepatic or renal impairment. Concurrent administration with certain antibiotics, i.e. clindamycin, erythromycin, troleandomycin, may result in higher serum levels of theophylline. Plasma prothrombin and factor V may increase, but any clinical effect is likely to be small. Metabolites of guaifenesin may contribute to increased urinary 5-hydroxyindoleacetic acid readings, when determined with nitrosonaphthol reagent. Safe use in pregnancy has not been established. Use in case of pregnancy only when clearly needed.

Adverse Reactions: Theophylline may exert some stimulating effect on the central nervous system. Its administration may cause local irritation of the gastric mucosa, with possible gastric discomfort, nausea, and vomiting. The frequency of adverse reactions is related to the serum theophylline level and is not usually a problem at serum theophylline levels below 20 μg/ml.

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NEW MEMBERS

The JOURNAL *takes this opportunity to welcome these new members into the Kansas Medical Society.*

Rodney Lee Bishop, M.D.
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Lawrence, KS 66044

Jaime T. Calderon, M.D.
7735 Washington Blvd.
Shawnee Mission, KS 66207

Richard Carlyle Carson, M.D.
Mt. Carmel Medical Center
Pittsburg, KS 66762

Luther Fry, M.D.
603 N. Fifth Street
Garden City, KS 67846

Captain King Gray, M.D.
Medical Arts Bldg. #3
Fourth & Maine
Lawrence, KS 66044

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UKSM-Kansas City
Kansas City, KS 66103

Thomas S. Harvey, M.D.
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Wichita, KS 67211

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Salina, KS 67401

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Hertzler Clinic
Halstead, KS 67056

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Fifth Annual UKSM—Wichita Issue

The Dean's Letter—1978

The University of Kansas School of Medicine–Wichita

D. CRAMER REED, M.D*., *Wichita*

Ed. Note: Vice Chancellor D. Cramer Reed, M.D., resigned effective July 1, 1978. Dr. Reed had directed the activities of the medical school in Wichita since 1973. He requested that warm regards and appreciation be extended to all members of the Kansas Medical Society for their support during the past few years.

THE PAST YEAR has brought closer to home the realization that adaptation and change can be both friend and foe. The University of Kansas School of Medicine-Wichita has experienced transition. A simple review of activities and accomplishments might convey our pride in our successes, but not the depth and fullness of our growth. We want to share with you what we have been doing, as well as our concerns and plans for the future.

Fiscal year 1978 has been a confusing time for many of our friends. First, certain changes in administrative structure were implemented in compliance with the request of the Liaison Committee on Medical Education. This involved the appointment of a dean for the Wichita campus of the medical school. It also prompted the assignment of a full time Vice Chancellor for Wichita. Associated with these changes, the Kansas State Board of Regents authorized a new title, The University of Kansas School of Medicine-Wichita (UKSM-W). There was yet another transition consisting of our move last July to the E. B. Allen Memorial Hospital.

As with any change, concerns were voiced in various corners. Just what would all of this mean? Fortunately, we can now look back in a positive way. Cooperation with the College of Health Related Professions at Wichita State has continued in a variety of ways. The support of UKSM-W by the Wichita professional community remains strong.

The growth of UKSM-W necessitated the move from Fairmount Towers on the WSU campus to our new location. What were formerly operating, obstetrical and patient care rooms are now offices, classrooms, and work areas. It's beginning to feel more like home. It now appears we will stay here for awhile although the lack of space for new faculty, more classrooms, and an auditorium is beginning to be felt.

Amidst these changes, however, an innovative faculty and staff assured continuity in medical education and the initiation of some exciting new programs. Our academic departments continue to be actively involved in faculty and curriculum development and improvement, expansion of clinical training opportunities, and development of community service projects.

In Surgery, a newly-developed subspecialty rotation in urology received enthusiastic responses from student evaluations. The Department of Obstetrics-Gynecology, in conjunction with Wesley Medical Center, has continued active participation and expanded the obstetric role in the statewide perinatal project. The department has continued efforts to further develop curriculum and has expanded clinical outpatient facilities.

The Psychiatry undergraduate obligatory clerkship was increased to eight weeks as UKSM-W progressed to reach its full complement of 50 students per class. Sang O. Lee, M.D., Glodys St-Phard, M.D., and Phillip Woollcott, M.D., joined the Psychiatry faculty to meet increased teaching demands. William LeVine, M.D., assumed the position as Director of the Psychiatry Residency program which will have six residents at the end of each year. Monthly psychiatric colloquiums at Wichita hospitals provided interaction between guest speakers, faculty, residents and students.

In Pediatrics, the development of a Genetics and Birth Defects Program is underway. Sechin Cho, M.D., joined the faculty in April to establish the program, and a genetics lab at Wesley Medical Center has been identified. The Kansas Regional Diabetes Education and Research Center was also initiated. Patient and professional diabetes education classes are offered as a part of pediatric clinic activities, and expanded treatment services are planned for the future. The Pediatric Outreach Program, conducted by Vernon James, M.D., provides examinations for over 100 children—many with learning disabilities—in a series of 11 clinics in various Kan-

* Vice Chancellor, UKSM-Wichita, 1001 North Minnesota, Wichita, KS 66614

sas communities. However, future funding for this program is uncertain.

Internal Medicine activities encompassed outreach programs for residents in Liberal, Great Bend, Halstead, Manhattan, and Salina; selected preceptorships for medical students with general internists; elective rotations for advanced students in off-campus sites throughout the state; and regular courses and activities directed at continuing education for physicians.

In Family Medicine, support was continued for the Harper Project which provided an interdisciplinary team educational approach for 19 medical students and several allied health professions students. The Harper and Kinsley Seminar Series provided continuing education for area physicians, nurses, and other health professionals. As many as 50 medical students completed preceptorship rotations in non-urban areas with primary care physicians. The goals and objectives for this experience continue to be refined.

Another high point in the year occurred when two new city-wide community-based residency programs received approval from the Liaison Committee on Graduate Medical Education. UKSM-W in affiliation with Wichita community hospitals will offer residencies in anesthesiology, under the direction of R. H. Robinson, M.D., and pathology, under the direction of William Reals, M.D.

The Division of Postgraduate Education sponsored 16 major conferences in 1977-78 and 114 other seminars/workshops, offering 275 hours of AMA Category I Credit to 4,251 participants. In Wichita, 175 credit hours were offered and 100 hours were offered in rural areas. Last December, George Khoury, M.D., was named Assistant Dean of Continuing Medical Education.

Of course, student and academic support activities were also important. Students continued to participate in the Wichita Public Schools Health Project teaching fifth and sixth graders about the cardiovascular system. Students also participated in Rural Health Weekend, spending three days with a primary care physician in a non-urban area.

The Computers Assisting Medical Professions (CAMP) Project placed a pilot terminal in Harper for use by students and faculty in that community as well as area health professionals. The project provides access to computer assisted instruction programs at Ohio State University Medical School and Massachusetts General Hospital. The Microwave Television Project (MEDWAVE) is nearing installation and will have the capability to link Wichita medical facilities. The system will be used to communicate seminars, grand rounds, and other instructional conferences. Publication of a monthly calendar and a newsletter, *The Monitor*, keeps faculty, staff, students, alumni, and others informed of all our activities.

The growth of the medical school has led to the establishment of our own central supply, print shop, and computer services. Computer Services has been used to establish a physician register and provide statistical application for the Department of Biometry, housed on the Kansas City campus. Other projects are in the mill for next year.

In addition to our primary objective of providing quality education, it is still our strong conviction that, as a community-based medical school, we must be responsive to community and outreach service needs. Dean Kortge, the former Director of Family and Community Medicine and Coordinator of the Preceptorship Program, was reassigned to assume the position of Associate Director of Health care Outreach. Working under the direction of Joseph Meek, M.D., Director of Health Care Outreach at The University of Kansas College of Health Sciences in Kansas City, Dean has been actively involved in working with numerous Kansas communities on a variety of health care concerns.

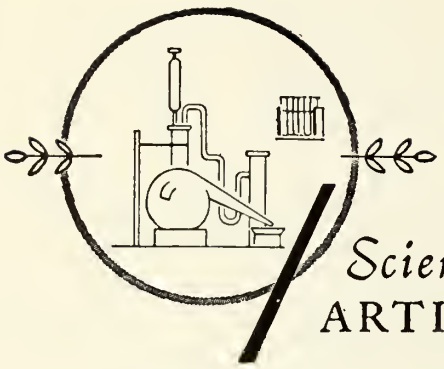
In coordination with the Health Systems Agency of Southeast Kansas, the Division of Health Care Outreach was involved in 19 community meetings concerning federal planning for health care. In a similar program designed to assess physician manpower needs, the staff traveled over 5,000 miles to meet with 350 people in 29 community meetings. Forty other community visits to address health concerns were also made.

Other outreach programs include the Hypertension Screening Program that screened over 3,000 people in Wichita, and the Mobile In-Service Education Program that provided continuing education to over 1,800 nurses and hospital employees. Seven new hospitals were added to the program last year.

In June, a series of 12 community workshops were conducted as part of the Rural Health Initiative Seminar Series. Outreach staff met with community leaders to discuss changing patterns of primary care medicine and different ways of recruiting health care professionals. More ways to meet the needs of health professionals and citizens in areas outside Wichita are being investigated and new plans are being formulated for the year ahead.

One area of concern and interest during the year ahead will be the further development of clinical

(Continued on page 502)



Community Hospital Review

Infection Following Placement of Foley Catheters

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A comprehensive study is reviewed indicating that catheter-associated urinary tract infections can be greatly reduced by utilization of appropriate infection control procedures by hospital personnel.

CATHETER-ASSOCIATED urinary tract infections remain the most common type of hospital acquired infection, accounting for more than 30 per cent of all reported nosocomial infection. Although it is unlikely that all such infections can be prevented, they can be minimized if nurses, physicians, and other hospital personnel utilize appropriate infection control procedures. Despite recent recommendations and guidelines for prevention of catheter associated urinary tract infections,[1] over 20 per cent of patients receiving closed sterile urinary drainage continue to acquire bacteriuria within the first five days of catheterization.[2] In most patients, it is assumed that catheter associated bacteriuria is benign and will resolve after removal of the catheter, or after catheter removal and antibiotic therapy. However, data collected over a three-year period from approximately 75 hospitals reporting to the Center for Disease Control show the rates of nosocomial gram negative rod bacteremias secondary to urinary tract infection are twice as high as rates of gram negative rod bacteremias secondary to infection at other sites.[3]

For the past several years, all patients undergoing Foley catheterization of the urinary tract at a community hospital have been placed on a bacteriologic monitoring protocol. In analyzing infection rates at this hospital, it became apparent that the proportion of infections related to the urinary tract was more than one-third higher than that reported nationally by hospitals of comparable size. Review of this data by the Infection Control Committee led to speculation as to the actual risks of infection following Foley catheterization in this hospital and, furthermore, whether a team approach to catheter care could reduce these risks. In order to answer these questions, the authors undertook a prospective surveillance of patients undergoing Foley catheterization during July, August, and September 1976, and correlated the data collected with the results of the Foley monitoring program. This report presents some of the results and conclusions derived from that study.

Materials and Methods

Routine Foley catheter bacteriologic monitoring has been performed at this community hospital since

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1969. An initial urine culture is taken on each patient at the time of catheterization. A sample of urine for culture is removed from the catheter tubing each subsequent day without breaking the closed system. The culture report sheet is generated each day and is placed on the patient's chart. Any growth on any of the cultured plates is identified and the attending physician is notified by means of the daily computer general report sheet of the presence of bacteria, the identification of the infecting organism, and the antibiotics to which the organism is sensitive. The daily monitoring continues until the catheter is removed.

During the months of July, August and September 1976, data were gathered in a prospective manner on all patients undergoing insertion of an indwelling Foley catheter. This data included information on hospital floor, medical service, date of admission, date of catheter placement, catheter inserter, and date of catheter removal. Additional information included whether the patient was receiving an antibiotic at the time of catheter insertion. Patients who subsequently developed bacteriuria were studied to determine the number of days of catheterization before bacteriuria, appropriateness of the antibiotic given, and the outcome of the infection. Infection in this study is defined as 10^2 organisms or greater, based on the rationale and methodology of Kunin[2] and Garibaldi.[4] Symptoms of bacteriuria were defined as the presence of fever, or any record made in the chart of complaints of suprapubic pain by the patient. All bacteria isolated underwent antibiotic sensitivity testing using the Kirby-Bauer disc method. If antibiotic was administered, review of the susceptibility data indicated whether or not the antibiotic given was one to which the organism was sensitive. Catheters were inserted by a group trained to do female catheterizations (SPS), male catheterizations (CS), operating room personnel, delivery room personnel, or the floor personnel depending on the sex of the patients and their location at the time of catheterization. All patients who had more than 10^5 organisms/ml of urine at the time of catheterization or who were catheterized less than 24 hrs, were deleted from the study.

Results and Discussion

During the three-month study period, indwelling catheters were inserted in 11.5 per cent of hospital admissions. Of the 1,171 patients studied, 353 (30%) had sterile urine upon catheterization, but subsequently acquired bacteriuria with at least 100 colonies/ml. Mean duration of catheterization was 3.6 days, and mean duration from insertion to infection was 4.1 days. *Table I* gives some comparative demographic data regarding the patients studied. There was a statistically significant difference between the mean age of 55 for the infected group, and the mean age of 42 for the non-infected group. The difference in the female : male ratio of 3.8 : 1 in those infected versus 3.2 : 1 in those not infected was not statistically significant. For those infected, average duration of catheterization was 6.7 days versus 2.5 days for those not infected, a difference with p value of $<.05$. Ten infected patients experienced an episode of gram negative sepsis as evidenced by positive blood cultures. This was 50 per cent of all gram negative sepsis noted in the hospital during the three-month study period. No evidence of gram negative sepsis occurred in non-infected groups. Mortality was significantly higher in the infected group with a 9.1 per cent death rate as opposed to 2.3 per cent for the non-infected patients. Infection in this study was defined as bacteriuria of 10^2 organisms or more, based on the methodology of Kunin[2] and Garibaldi.[4] The rationale of this approach is that of the catheterized patients who develop 10^2 colonies/ml, over 90 per cent develop 10^5 colonies/ml or more if the catheter is left in place and no antibiotic is administered.

Review of the overall infection rates for all nursing units with over 30 patients catheterized indicated that several units had infection rates higher than the average of 30 per cent. The differences were statistically significant at $p<.05$.

Overall infection rates were also related to hospital services. Two services (medicine, 57% and orthopedics, 63%) had infection rates significantly higher than the mean of the whole study population ($p<.05$).

A comparison of infection rates among patients as defined by the five groups responsible for catheter insertion showed patients catheterized by special services personnel had an infection rate of almost 50 per cent greater than did patients catheterized by central services technicians. Patients catheterized in

TABLE I

CHARACTERISTICS OF THE PATIENT POPULATION

Characteristics	*Infected*	*Non-Infected*	*Total*
Mean age	55	42	46
Female/male ratio	3:8	3:2	3:4
Mean catheter duration	6.74	2.51	3.64
Sepsis, GNR	10	0	10
Mortality	9.1%	2.3%	4.3%

TABLE II

FREQUENCY DISTRIBUTION OF 443 BACTERIAL ISOLATES RECOVERED FROM 364 PATIENTS

Organism	*Isolates* NO.	%	*Cause of Infections* %
E. Coli	161	(36)	44
K. Pneumoniae	46	(11)	13
Proteus	38	(8)	10
Enterobacter	25	(6)	7
Pseudomonas	29	(7)	8
Other gram neg.	28	(6)	8
Staphylococcus epi.	43	(10)	12
Enterococcus	45	(10)	12
Other gram pos.	28	(6)	8
	443	(100)	

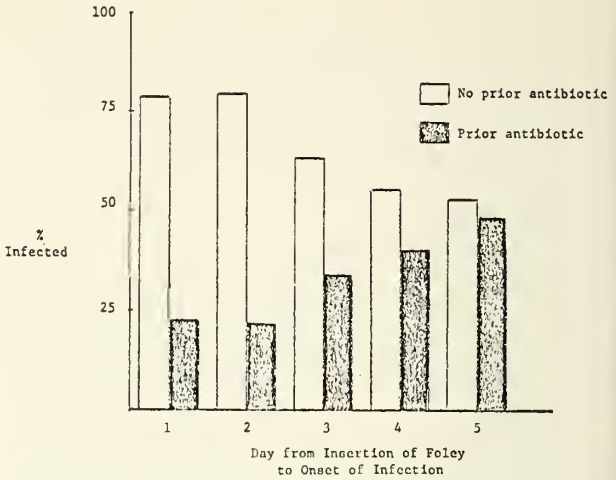


Figure 1. Duration from insertion to infection onset in patients with and without antibiotics.

the delivery and operating room had significantly lower rates of infection.

Table II illustrates the frequency distribution of the 443 bacterial isolates recovered from 353 patients. *E. coli* was the most common organism isolated and occurred in 44 per cent of the infected patients. It was found in 36 per cent of the isolates. Of interest, pseudomonas occurred in only 7 per cent of the isolates and in 8 per cent of the infected patients. Although only 20 per cent of these infections were symptomatic by the criteria defined, 182 patients (50%) received antibiotic treatment.

Considering these 182 patients, 146 (80%) received antibiotics to which the organisms were susceptible in vitro by the Kirby-Bauer disc test. Thirty-six (20%) received drugs to which the organism was resistant. Of those patients treated after the onset of their bacteriuria, 76 per cent received one drug, 21 per cent two drugs, and 3 per cent three drugs. Of the patients receiving antibiotics prior to infection, 72 per cent were treated with one drug, 22 per cent with two drugs, and 6 per cent with three drugs. Five per cent of the infected patients received either ampicillin or cephalothin, and 18 per cent received an aminoglycoside. Thus, the occurrence of these infections resulted in significant cost and potential drug toxicity for a significant number of patients in addition to the ten instances of gram negative sepsis referred to above.

Figure 1 shows the distribution pattern of the 232 patients infected in the first five days following catheterization. This distribution of infections is depicted according to day post-catheterization at which bacteriuria was detected in those receiving and not receiving concomitant antibiotics. The significantly increased proportion ($p < .02$) of the infections occurring in the first three days was present in those not receiving concomitant antibiotics. However, by the fifth day of catheterization, the distinction of bacteriuria between these two groups was approximately equal. In addition, when the organism frequency distribution was analyzed, 69 per cent of the isolates of pseudomonas occurred in the prior antibiotic group, whereas 68 per cent of the *E. coli* isolates were obtained from the urine of patients not receiving antibiotics prior to or concomitant with catheterization. This difference was highly significant with p value $< .001$. Although the frequency distribution of other organisms did not differ significantly when patients with prior versus no antibiotics were compared, we interpret the shift of organisms from *E. coli* to pseudomonas to represent the antibiotic selection of more resistant flora in the patients affected.

The preceding data appear to indicate significant differences in overall infection rate depending upon the patient's location in the hospital, medical service required, and identity of the team placing the catheter. The question is, are these real or only apparent differences. Pertinent to this question is the identification of compounding variables that might make non-comparable the overall rates of infection in these groups. In addition to the effect of prior and concurrent antibiotic use already described, the duration of catheterization is a variable that must be standardized and so, after the method of Kunin, we re-examined our data looking at the cumulative (rather than overall) risk of urinary tract infection. This analysis assumes that patients becoming infected will remain so, and should be counted in the denominator each day if they remain catheterized. On the other hand, patients whose catheters are removed when the urine is sterile prior to the day under consideration are not counted in the denominator, as

TABLE III

DAILY CUMULATIVE RISK OF URINARY TRACT INFECTION AT 1, 3, AND 5 DAYS POST CATHETERIZATION BY NURSING UNIT

Category NURSING UNIT CODE	*AR**	*Cumulative Risk of Catheter Associated UTI* DAY 1 No.	%	DAY 3 No.	%	DAY 5 No.	%
12	52	7/40	18	17/33	52	18/27	67
22	59	4/31	13	8/24	33	12/21	57
24	10	8/160	5	14/26	54	16/18	89
4	62	7/38	18	18/31	58	20/26	77
6	37	3/40	8	13/27	48	14/19	74
8	21	2/82	2	12/56	21	14/30	47
2	34	6/112	5	26/75	36	31/55	56
32	30	6/63	9	17/48	35	19/31	61
34	48	4/26	15	9/18	50	12/14	75
14	24	14/266	5	54/115	47	57/96	60

* Attack Rate: Overall % of patients developing UTI following Foley catheterization.

they are not at risk for the duration specified.

Table III illustrates this cumulative risk analysis for various subsets of the patient population with initial sterile urine. Here we see the category "Nursing unit" with the overall attack rate indicated in the second column and the cumulative risk as determined by the numerator and denominator indicated for days one, three, and five following catheterization. Notice that although the overall infection rate, as indicated previously, varied from 10-62 per cent in the units shown here, the corresponding cumulative infection rate on day 3 was significantly lower only for #8 (Nursing unit code), and when day 5 is considered, the difference between #8 and the other units has disappeared. Perhaps more striking is #24, with an overall attack rate of 10 per cent but a cumulative rate of 54 per cent by day 3, and 89 per cent by day 5, the highest rate noted for any of the units studied. Thus, although significant differences in infection rate following catheterization were noted between nursing units on day 1 following catheterization, by days 3 and 5 these differences were not as dramatic and probably reflect differences in the age, sex, and underlying disease of the patients.

Figure 2 illustrates the effect the sex of the patient had on the cumulative infection rate where the bias of catheter duration has been eliminated. The descending lines indicate patients at risk; the ascending lines show cumulative infection rate. In contrast to the overall analysis which failed to show a significant difference between males and females regarding rate of infection, here we can see by day 3 the risk of infection was twice as great in females, and by day 5 remained one third higher. Overall infection rates were comparable between the two sexes because, as can be seen by the descending lines indicating per cent of patients still catheterized, rates of catheter removal in females within 72 hours were over twice that of males, with less than 50 per cent of the catheters remaining in female patients by day 3.

Table IV shows cumulative infection rates by service and catheter inserter. Again the same pattern is apparent. Statistically significant overall differences in infection rates noted in the first column and shown previously in this report are largely negated by day 5, when cumulative risk analysis is used. For example, the Gyn service with an overall rate of 22 compared to medicine with an overall rate of 57 shows a comparable infection risk by day 3, and this trend continues on day 5. Although infections on urology would appear to be lower with this analysis when the sex and prior antibiotic usage of patients is considered, clinical importance of these differences is difficult to assess. Finally, in considering the catheter insertion team, although the cumulative risk analysis would tend to implicate catheters placed by SPS, CS and the "others" group, inclusion of data regarding sex, age, and underlying disease in the analysis makes these statistical differences clinically suspect. However, all these subgroups show high risk of infection for the duration of catheterization considered. Perhaps a more useful way to put perspective on this data is to compare it with two similar studies described in the literature; the first was performed by Calvin Kunin and associates at the University of Virginia School of Medicine in the early 1960s, and the other reported by Richard Garibaldi from the Salt Lake City Latter Day Saints Hospital in 1974. *Figure 3* depicts comparative data as determined by the

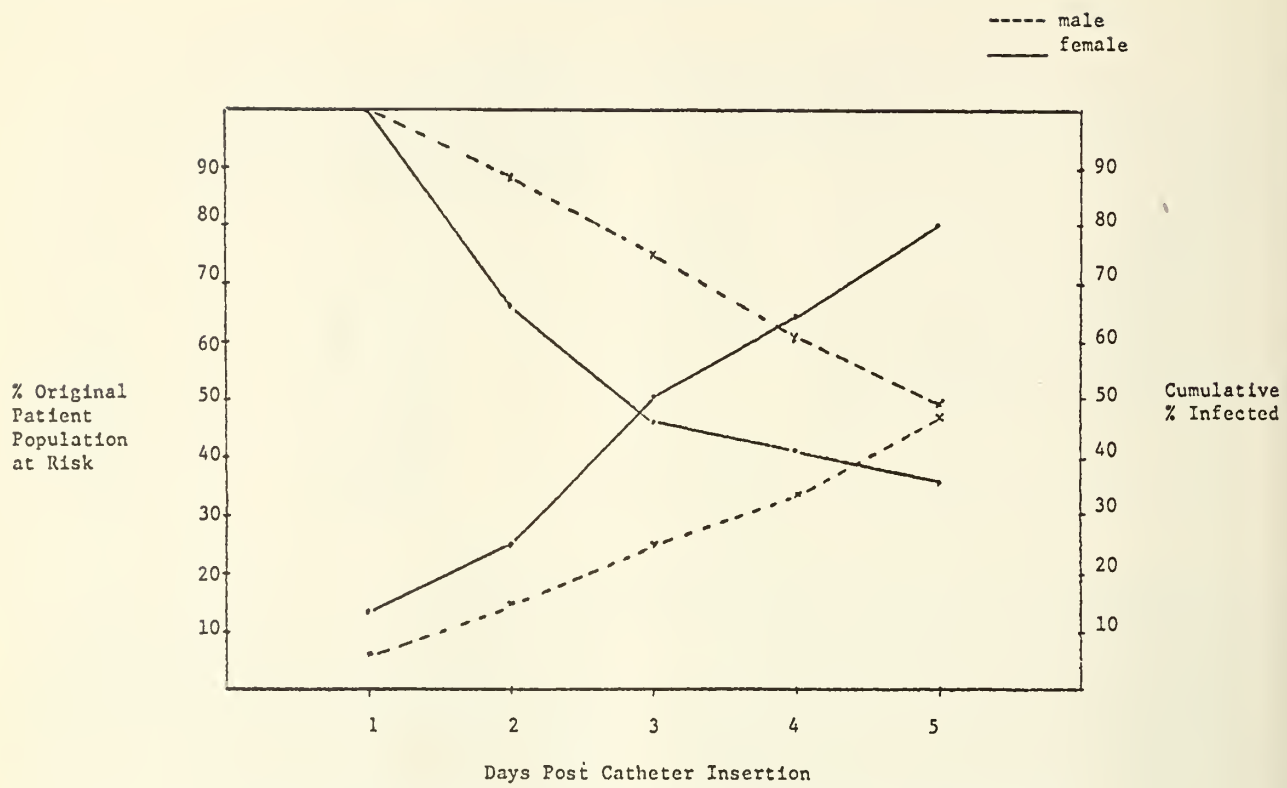


Figure 2. Cumulative infection rates for patients at risk for 5 days of study.

TABLE IV

DAILY CUMULATIVE RISK OF URINARY TRACT INFECTION AT 1, 3, AND 5 DAYS POST CATHETERIZATION BY MEDICAL SERVICE AND CATHETER INSERTION

Category		*Cumulative Risk of Catheter Associated UTI*					
		DAY 1		DAY 3		DAY 5	
SERVICE	*AR**	No.	%	No.	%	No.	%
Med	57	21/123	17	52/100	52	66/86	77
Surg	26	15/236	6	44/159	28	51/96	53
Gyn	22	22/478	5	92/180	51	109/133	82
Ortho	63	8/44	18	19/39	49	23/35	66
Urol	24	1/42	2	7/30	23	8/17	47
INSERTER	*AR**	No.	%	No.	%	No.	%
SPS	51	28/182	15	72/115	63	85/101	84
CS	49	6/85	7	23/70	33	30/55	54
DR	10	5/125	4	12/23	52	14/15	93
OR	20	14/430	3	74/231	32	88/144	61
Others	44	16/105	15	34/69	49	40/52	77

* Attack Rate: Overall % of patients developing UTI following Foley catheterization.

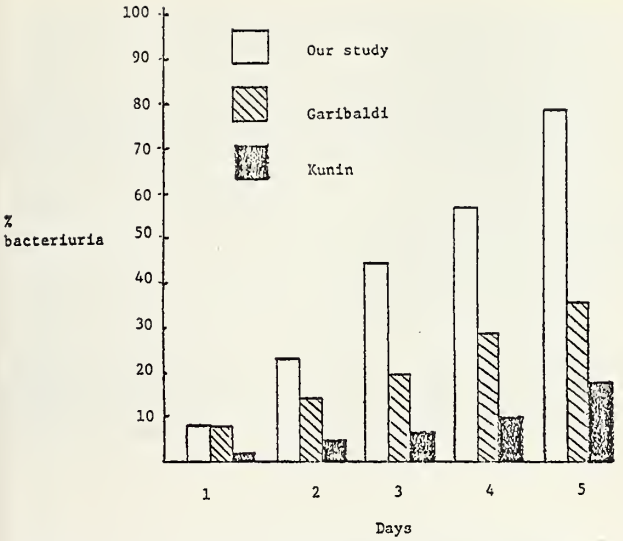


Figure 3. Daily cumulative risk of catheter associated UTI—A comparison of three studies.

cumulative risk approach. The differences are obviously substantial.

It appears that the differences reported here are largely a reflection of differences in the methodology of the studies compared. Kunin's group was studying the use of a closed drainage urinary tract catheterization system, and nursing and ancillary personnel were intensively instructed "via a series of meetings with the hospital infections committee." The maintenance of the closed system was intensively surveyed. Only an occasional break in technique was observed. On the other hand, Garibaldi, working in a 600-bed community hospital, was primarily studying different types of closed drainage equipment, and hospital personnel were not aware that the study would involve an analysis of catheter care techniques as well. Thus, although personnel were instructed in the use of several systems of closed drainage, maintenance of closed drainage was not extraordinarily emphasized as it was in Kunin's study. Garibaldi's group observed a multitude of errors in managing the closed drainage catheter system, including evidence of episodic disconnection of the catheter tubing junction 27 per cent of the time and improper positioning of the drainage bag 21 per cent of the time. These errors were associated with the subsequent development of bacteriuria in the patient. During our study, no efforts beyond routine inservice training were made to specifically educate nursing personnel regarding maintenance of the closed drainage system or catheter care technique; in fact, since the Foley monitoring program had been routine for four years and patient data was collected by nurse epidemiologists, there was no indication that a study of urinary tract infection following Foley catheterization was being conducted.

The exceptionally high cumulative risk data in this study appears to reflect more nearly the routine care of the Foley catheter system and the risk of associated infection which exists in the absence of efforts such as those of Kunin and Garibaldi. Furthermore, the comparison of these data imply that an infection control program which could achieve an intensity of inservice education and catheter care comparable to that achieved by Kunin and Garibaldi would have significant impact on the overall risk of nosocomially acquired urinary tract infection following Foley catheterization.

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Breast Surgery

Current Concepts in Augmentation

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THE CONTOUR of the breast is universally accepted as a characteristic feature of the adult female and symbolizes a mature woman. When insufficient contour is present, adequate contour has been sought through use of special garments, exercise, diets, and medications that claim to facilitate achievement of this goal. Prior to 1950, the surgical enlargement of the breast was done by free grafts of dermis, fat, and fascia. The early results were good, but with time, so much shrinkage occurred that free grafts were proven unsatisfactory. An active search began in the last part of the 19th century for a synthetic material suitable for augmentation. Most of the substances tried were injectable hydrocarbons that were inflammatory and very unsatisfactory. In 1951, the first clinical report on the use of polyvinyl alcohol (Ivalan) led to the use of that substance as the primary material for augmentation.[1] However, its porous nature led to an ingrowth of fibrous tissue resulting in unnatural hardness. The development of silicone in the 1960s progressed to the manufacture of a silicone gel that is nonporous and of soft consistency. This silicone material has been used almost exclusively in augmentation mammoplasty for the past 15 years, and the surgical procedure of augmentation mammoplasty has become one of the most frequently performed cosmetic operations in the world.[2] It is estimated that approximately 80,000 augmentations are performed annually in the United States.

Material Studied

This report is limited to 300 consecutive cases of augmentation mammoplasty for aplasia, hypoplasia, post partum involution, and asymmetry of the breast. Other plastic surgery of the breast — such as mastopexy for ptosis without augmentation, reduction mammoplasty for macromastia, nipple inversion, subcutaneous mastectomy with implants, and reconstruction after simple and radical mastectomy — will be reported in a later study. The most recent 300 augmentation mammoplasties performed by the authors have been reviewed to assess the type of patients operated, and the surgery has been studied from a standpoint of results and complications.

Surgical augmentation of the breast has progressed from free dermal fat grafts to insertion of inert silicone implants. Current procedure is outlined and 300 surgical cases reviewed delineating the physical and social background of the women seeking surgery. Assessment and postoperative findings are reported.

The Patient

It is interesting to review augmentation cases to determine what kind of woman seeks surgery. The augmentation population of the Kansas area appears similar to that reported in other areas of the country. The average age in our study was 32 years. The oldest patient was age 62 years and had recently undergone a face lift; she requested augmentationn to improve breasts that had become ptotic with time and postpartum involution. The youngest patient was age 14 years with absence of one breast. She had been followed since age 12 years with no change in the size of the breast. Married women represented 75 per cent of the group. The numbers of single and divorced were equal with 12 per cent each, and 1 per cent were widowed. The most common patient to seek augmentation mammoplasty is happily married, in her early 30s, with breasts diminished by postpartum involution. These are similar to the findings in other series.

Preoperative Findings

Fifty per cent of the patients in this series gave a history of larger breasts in the past; the breasts invo-

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Figure 1

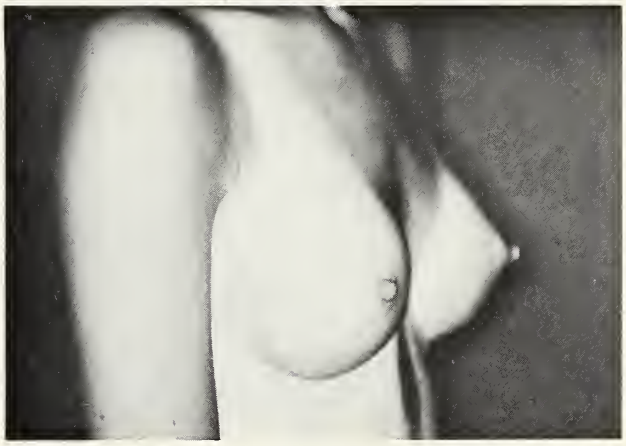


Figure 2

Figures 1 and 2. Preoperative case of hypomastia and the postsurgical augmentation mammoplasty results at 3 mos.

luted after pregnancy or nursing. Only about 10 per cent of the patients in this group had developed striae of the skin during enlargement with pregnancy. Ptosis existed in 15 per cent of the patients; this was only mild ptosis in which the nipple did not fall below the inframammary line when the patient was upright. If the ptosis is great enough for the nipple to be below the inframammary line, a mastopexy is required, and this existed in only 1 per cent of the cases.

Ten per cent of the patients had a significant degree of asymmetry. Some had total absence of one breast. The most common reason for this was biopsy of the breast during infancy. Two of the patients requiring unilateral augmentation also underwent a contralateral reduction mammoplasty as the opposite breast was too large to achieve a symmetrical result. One to two per cent of the patients gave a history of asymmetry due to previous breast surgery.

Psychiatric Considerations

While psychiatric studies have been conducted regarding this surgery, it appears that little more has been accomplished than would be expected with any significant change in physical appearance.[3] Patients in general feel more feminine in appearance, and this does frequently influence their lives. It does not in any way alleviate other associated anxieties or correct other self-image problems. The augmentation mammoplasty is of great help to young women with severe asymmetry. This problem can seldom be successfully disguised by ordinary clothing, and is impossible with many sports clothes. This can be a chronic source of embarrassment, and the treatment is correction of the physical deformity.[4] Two patients had postoperative psychiatric problems which are discussed under complications.

The Implant

The implants now in use consist of a thin shell made of a pliable silicone material filled with a viscous silicone gel or inflated with normal saline. There has been little problem with leakage or rupture of the implants filled with silicone gel, but leakage has occurred in saline-filled prostheses with reports of up to 5 per cent of patients experiencing problems with loss of volume.[5] Both types of implants are available for problems such as asymmetry due to rib cage deformity. Ninety-eight per cent of patients in this series received silicone gel prostheses, and 2 per cent received saline inflatable type.

Injectable silicone has not been approved by the Food and Drug Administration for clinical use. The manufacturers and investigators of injectable silicone have never advocated its use for breast augmentation. Injectable silicone has been imported from foreign countries and used for breast augmentation, but this frequently results in the formation of lumps; it is always dangerous, and at present is illegal.

Method of Surgery

Ninety-two per cent of the augmentation patients chose outpatient surgery due to the convenience and decreased expense. The most common reason cited by the 8 per cent who elected to have their surgery in the hospital was combination with other surgery, treatment, or diagnostic procedures. Since establishment of operating rooms in the office, 70 per cent



Figure 3



Figure 4

Figures 3 and 4. Preoperative case of breast asymmetry and the postoperative correction by a right unilateral reduction mammoplasty and left unilateral augmentation mammoplasty.

of patients have elected to have this surgery performed there under a local anesthesia.

The laboratory specimens are obtained in an accredited outpatient laboratory 48 hours prior to the scheduled surgery. Complete blood count, urinalysis with microscopic examination, and partial thromboplastin time are the minimum routine laboratory findings obtained on each patient. The patient is instructed to shower or bathe well on the morning of surgery. On admission, clothing is changed to a completely disposable operating gown and shoes, to minimize contamination of the patient and the operating room.

Prior to sedating the patient, the cautery ground plate is secured; and ECK monitor pads, finger pulse for the cathode ray display, and the pressure sensing light display blood pressure cuff are attached. An IV of 5 per cent dextrose and normal saline is utilized to administer intravenous medication. Intravenous meperidine and Valium are administered in dosages sufficient to induce a feeling of drowsiness and euphoria and to provide analgesia during the period of time the local anesthetic is injected. This usually requires 100 mg of meperidine and 10 mg of Valium.

The patient then undergoes a five-minute skin preparation with Betadine solution and is draped in a sterile field with only the anterior chest wall exposed. Lidocaine solution 0.5 per cent with epinepherine 1 : 200,000 is used for the local anesthesia, total dosage not to exceed 400 mg of lidocaine. A 12-cm circular area between the breast and pectoralis muscle is infiltrated with the lidocaine and epinepherine.

Through the 5-cm inframammary skin incision the breast is elevated from the pectoralis muscle far enough to accommodate insertion of the Silastic implant. A fibroptic retractor provides light for the submammary dissection and the localization and electric cauterization of bleeding vessels. The Silastic implant of appropriate size and shape is selected and inserted; the breast and subcuticular tissue is closed with 5-0 polypropal glycolic acid suture; and the skin is closed with 6-0 nylon suture.

The only dressing used is a strip of micropore tape over the sutures and a brassiere of appropriate size to hold the implants in correct position. The patient wears this brassiere at all times for one week except when bathing. The sutures are removed on the seventh postoperative day.

Postoperative pain is mild, and Tylenol 800 mg with 30 mg of codeine is usually all that is required for satisfactory postoperative analgesia.

Complications

Hematoma formation is the most common problem to arise in the early postoperative period. Seven per cent of the patients in this series required an exploration of the operative area to control bleeding. This most frequently occurs during the first six hours following surgery. It does not appear to affect the long term results. Infection occurred in four patients. This problem requires the removal of the implant for healing, and the implant is reinserted at a later date.

Capsule formation around the implant has not been a serious problem, but it is a disturbing one as the thickening of the fibrous capsule gives a less than ideal result. More attention has been directed toward this finding than any other during recent years. The formation of a layer of fibrous tissue around the implant occurs in all cases as this is the normal

response of the body to the presence of the silicone material. In some cases, this capsule becomes thicker and firmer, and is quite often unilateral. If the fibrous capsule around the implant is interrupted, the breast will again resume a normal consistency. This can be accomplished by surgery, as was done in 11 cases in this series, or by a closed capsulotomy, as was performed 69 times. A closed capsulotomy involves rupturing of the fibrous capsule surrounding the implant by manual compression of the breast. This procedure is done on a routine office visit.[6] While this method is usually effective in restoring the breasts to normal consistency, we do not know at present how permanent these results will be.

Two patients encountered postoperative psychiatric problems. Both had been under psychiatric treatment in the past, and each had been referred by her therapist with a recommendation for surgery.

One case presented a postoperative hysteria at one week, in which the patient claimed she was abnormally large and wanted the implants removed immediately. There was no doubt the breasts were quite swollen and unusually ecchymotic, and it was difficult to convince the patient her condition was temporary. She has since indicated her satisfaction with the final result.

The second patient was apparently happy with the result for the first year, and then returned claiming she was embarrassed by the size of her breasts and wanted the implants removed. She wore size 32-B brassiere. An attempt was made to return her to her therapist but she refused, and at the insistence of herself and her husband the implants were removed. One week following removal she requested replacement of the implants. Additional surgery was refused until such time as full psychiatric evaluation was obtained. The patient refused this, and has not been heard from since.

Routine psychiatric evaluation is not part of the preoperative work-up, but has been requested when the history indicates the advisability of obtaining more information regarding the patient's motivation for surgery.

Discussion

The infrequent occurrence of complications, and the fact that most of these complications when they occur are not serious, has made augmentation mammoplasty a very satisfactory procedure during the last two decades.[7] The patient level of satisfaction with the surgery is high.

The surgery does not interfere with the detection or treatment of breast cancer. Normal breast tissue is

(Continued on page 519)

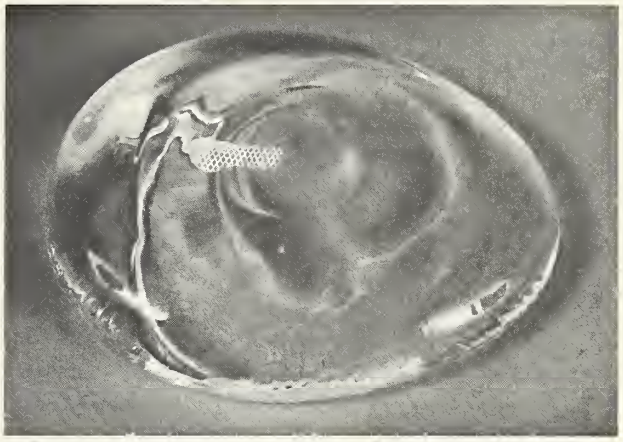


Figure 5



Figure 6

Figures 5 and 6. Silastic implant filled with Silastic gel. Soft pliability can be seen when implant is held between the fingers.

Swan-Ganz

The Rational Use of Pulmonary Artery Catheterization

PETER A. CONRARDY, M.D.,* *Wichita*

INSERTION of a pulmonary artery catheter, the Swan-Ganz catheter, is an invasive technique requiring the insertion of a large catheter (5-7 French) into a central vein. The catheter passes through two heart valves (tricuspid and pulmonic), coming to rest in the pulmonary artery. Use of the Swan-Ganz (S-G) catheter predisposes the most critically ill patient to new and more terrifying complications. Despite this, the S-G catheter is now recognized as one of the most reliable and useful monitors of the low output syndrome (shock).

During insertion of the S-G catheter, useful clinical data may be obtained by observing the pressure wave forms and noting the pressures as the catheter is advanced to the right atrium, the right ventricle, and the pulmonary artery. For example, if all diastolic pressures are the same, we may suspect cardiac tamponade as the cause of the shock-state. The configuration of the wave forms can be useful in delineating valvular disease.

Observation of the pulmonary artery, systolic and diastolic pressures, and the pulmonary capillary wedge pressures (PCW) has exposed the limitations of the central venous pressure (CVP) monitor in the critically ill patient.[1, 2]

Physiologically, the heart functions as two pumps. For many years, the right atrial pressure, as measured by the central venous catheter, was believed to reflect the filling pressure of these pumps, and in turn the stroke-work subsequently produced by each pump. However, subsequent analysis of independent right and left ventricular function curves has demonstrated that the shape and slope of these curves are different, and the stroke-work scales are also different.[3, 4] Assumptions based upon the relationship between the mean right atrial pressure and the left ventricular stroke-work are subject to error.[1, 5] Therefore, the cardiac output (left ventricular output), which is a function of left ventricular stroke-work, cannot be accurately evaluated unless the left ventricular filling pressure (mean left atrial pressure) is known. This principle was put forth by Frank, Straub, Wiggers, and Starling. This later became known as Starling's Law of the Heart, wherein Starling concluded that "the energy of contraction is a function of the length of the muscle fiber. The larger the diastolic volume of the heart (within physiologic limits), the greater is the energy of its contraction. . . ."[6] Clinically, myocardial fiber length is proportional to left ventricular end-diastolic volume, which in turn varies directly with the left ventricular end-diastolic pressure.

The Swan-Ganz catheter has afforded increased accuracy in assessment of the cardiovascular system. However, the data derived from its utilization requires sophisticated analysis to avoid complications and to assure accurate assessments. Ramifications of process and analysis are reviewed and explained.

In the absence of mitral valvular disease, mean left atrial pressure can be used to estimate left ventricular end-diastolic pressure. In a wide range of pressures and a number of disease states, pulmonary capillary wedge pressure can be used to reliably estimate left atrial pressure, whereas central venous pressure cannot[1, 2] *(Figure 1)*.

Variability of the CVP independent of the pulmonary capillary wedge pressure has been associated with acute myocardial infarction, decompensated cirrhosis, acute respiratory failure, severe peritonitis, and multisystem injuries. In any patient with one of these disease processes or any patient in whom the cause of the shock-state is unclear, the insertion of a Swan-Ganz catherer is indicated.

Technique

A number of techniques may be used to insert the S-G catheter. It may be inserted to the antecubital vein by cut-down. Percutaneous insertion at a sight other than the antecubital fossa is preferable, because the patients are not encumbered by the catheter and because patient movement is less likely to alter

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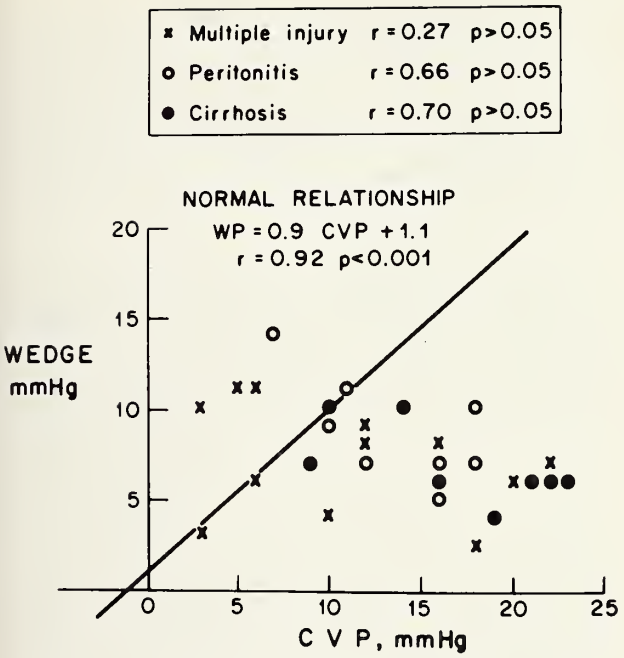


Figure 1. Pulmonary Capillary Wedge Pressure (PCW) is proportional to myocardial fiber length. In the absence of mitral valvular disease, PCW is a reliable estimate of left atrial pressure. (Used with permission of Dr. Joseph M. Civetta and J.Lippincott Co.)

the position of the catheter tip. Percutaneous puncture of a central venous vein is preferred, either through the subclavian, internal jugular, or femoral vein. The distance from the point of insertion of the S-G catheter to the level of the right atrium is dependent upon the site of insertion *(Table I)*. When the catheter tip is within the thorax, the balloon is inflated with an appropriate volume of air. The inflated balloon is caught up by the blood stream and swept through the atrium and tricuspid valves into the right ventricle up through the pulmonary valve into the pulmonary artery, until it wedges in a distal branch of the pulmonary artery. The pressure should be noted and recorded as it is passed through the various chambers of the heart. The balloon is then deflated. A chest x-ray should then be taken to verify the position of the catheter tip. Upon reinflation of the balloon, the forces of the flowing blood stream will again propel the catheter distally into a pulmonary artery until it wedges, as previously described.

Measurement of Pressures

In order to obtain accurate measurements of the pulmonary artery pressures (systolic and diastolic) and the pulmonary capillary wedge pressures, the diaphragm of the pressure transducer must be zeroed in at the "phlebostatic" point. The "phlebostatic" or "zero" level locates the right atrium. The

TABLE I

DISTANCE FROM THE POINT OF INSERTION OF THE S-G CATHETER TO THE LEVEL OF THE RIGHT ATRIUM

Rt. Antecubital Fossa	35-40 cm
Lt. Antecubital Fossa	45-50 cm
Internal Jugular Vein	10-15 cm
Subclavian Vein	10 cm
Femoral Vein	35-40 cm

"phlebostatic" point is at the midpoint of an antero-posterior line, drawn from the fourth interspace anteriorly to the back. Although this point is not believed to change with a change in posture, it is customary to locate this point and measure central venous and pulmonary artery pressures with the patient in the supine position at zero degrees. The pulmonary artery pressure trace should be continuously monitored on an oscilloscope in order to observe for, and immediately correct, inadvertent wedging of an S-G catheter. When the flow is interrupted by impaction of the distended balloon, the pressure is transmitted via a static column of blood to the next active circulatory bed — the pulmonary veins that are in direct connection with the left atrium. The pressure tracings obtained by the pulmonary artery catheter are subject to spurious oscillations due to catheter movement with the beating heart — usually at the rate of 20 cycles/second or greater. Because the important biological changes in the intravascular pressures in humans occur in less than 10 cycles/second (5-8 cps), the catheter manometer recorded systems deserve special attention in regard to optimal damping, to minimize this artifact. Assuming that there is no mitral valvular disease, and that respiratory oscillations are minimal, the mean pulmonary capillary wedge is the pressure measurement which is most clinically useful. If respiration, either spontaneous or mechanical, results in wide excursions of the PCW tracing, the PCW pressure should then be measured at a point coincident with end-exhalation. This point can only be identified by reading the PCW pressure from a calibrated oscilloscope which is displaying the pressure trace. Digital readouts are not sufficiently sensitive to measure the effect of transmitted respiratory intrathoracic pressures. If the patient is being treated with positive end-expiratory pressure (PEEP), this should be maintained during the readings while noting the difference when the PEEP is decreased to zero once every eight hours. This gradient is useful, and should be recorded on critical care nursing flow sheets.

Complications

The complications encountered in the use of the pulmonary artery catheter are, indeed, terrifying; however, they are preventable. The complications most frequently encountered are: (1) damage to the pulmonary parenchyma; (2) cardiac arrhythmias; (3) balloon rupture; (4) electrical induction of cardiac dysarrhythmias; (5) infection and thrombophlebitis; (6) knotting; (7) thrombotic endocardial vegetation; and (8) ventilator disconnect.

Damage to the Pulmonary Parenchyma

Pulmonary parenchymal damage is preventable by close observation and appropriate technique. This complication results from obstruction of the pulmonary artery blood flow due to peripheral migration of the pulmonary artery catheter to a wedged position. This obstruction to pulmonary artery blood flow is essentially a pulmonary embolism, and results in pulmonary infarction distal to the point of obstruction. Peripheral migration of the catheter occurs with each inflation of the balloon. After the S-G catheter is first inserted, there usually is a sizable trans-cardiac loop. With time, the catheter material softens and the trans-cardiac loop shortens with inflation of the balloon, resulting in further and further peripheral migration. For this reason, the pulmonary artery trace should always be continuously monitored. If a continuous pulmonary capillary wedge trace is observed, the catheter should be pulled back 1-2 cm at a time until the pulmonary artery trace is observed.[7]

Continuous monitoring of the pulmonary capillary wedge pressure for the above reasons should never be done; the pulmonary artery catheter should not be wedged for longer than one to two minutes.

Damage may also result from direct trauma of the catheter or the balloon. The normal pulmonary artery is a thin structure of limited tensile strength. Thus, when excessive amounts of air are used to inflate the balloon, or if saline is used to inflate the balloon, dangerously high lateral wall pressures can result in parenchymal damage or even rupture of the pulmonary artery.[8] This occurs: (1) if the allowable volume of air is used for wedging, even though the air required to produce a wedge trace is significantly less than the total allowable volume; (2) if saline is used to inflate the balloon; (3) in patients who are anticoagulated; and (4) in patients with serious pulmonary hypertension.

Damage to the pulmonary parenchyma can be prevented by nursing awareness, with continuous monitoring of the pulmonary artery pressure trace. The catheter should be retracted when the air required to produce the wedge trace is substantially less than the total allowable (0.8 cc for 5 French, and 1.5 cc for 7 French). The balloon should be filled incrementally, 0.2 cc at a time, while observing a change in the pressure trace from one indicative of the pulmonary artery to one indicative of the pulmonary capillary wedge pressure.

Cardiac Arrhythmias

Ventricular fibrillation and episodes of ventricular tachycardia have been reported in patients who were being monitored with an S-G catheter. However, the incidence of these catastrophic arrhythmias is far less than has been reported to occur with conventional cardiac catheters (without balloon).[9] These arrhythmias have occurred in patients who have documented myocardial ventricular irritability, and when the catheter tip is displaced from the main pulmonary artery to the pulmonary outflow tract. The catheter tip stimulates the sensitive endocardium of the right ventricular outflow tract resulting in serious arrhythmias. This can be prevented by inflating the balloon in order to cushion the impact of the catheter. A defibrillator and arrhythmia drugs must be available in the patient care areas.[10]

Balloon Rupture

This complication is of little consequence in the absence of congenital heart disease. In patients without congenital myocardial defects, 0.8-1.5 cc of air has no apparent deleterious effect.[9] Balloon rupture is due to excessive volumes of air used for inflation of the balloon, and to the repeated use of the same S-G catheter on different patients. The only possible severe consequence of balloon rupture is fragmentation and subsequent embolization of the balloon fragment.

Electrical Induction of Cardiac Dysarrhythmias

This is due to leakage currents from poorly isolated electrical or electronic devices. These leakage currents pass through the fluid column (low impedance) pathways, *i.e.* the central venous pressure or S-G catheter. The use of appropriately isolated electronic devices in areas where saline filled catheters are placed close to the heart is an important preventive measure.[11]

Infection and Thrombophlebitis

Manipulation of the catheter and poor skin sterilization may result in infection and thrombophlebitis.

Appropriate sterile technique should always be used when the S-G catheter is passed. Percutaneous insertion in a central vein is recommended at the level of the subclavian or internal jugular, since wide incisions are not used and since the catheter can be relatively immobilized.[12]

Knotting

This has been seen only with the #5 French S-G catheter. It occurs when the S-G catheter has been passed beyond the point when one would expect the catheter to be within the pulmonary artery, but is not, as indicated by the pressure trace. It most commonly occurs in patients with a dilated heart due to congestive heart failure.[13]

Knotting of the S-G catheter can be prevented by using an image intensifier during insertion of the S-G catheter, or by using a #7 French S-G catheter in patients with cardiomegaly. When not using an image intensifier during insertion, refrain from inserting the catheter 10-20 cm beyond the point you should expect the PA trace.

Thrombotic Endocardial Vegetation

The incidence of non-bacterial endocarditis in patients who have been catheterized with an S-G catheter is significantly greater than in those who were not catheterized. In a series of 88 patients, 3 with valvular lesions had the S-G catheter in place for a mean of 5 + 1.6 days, while the mean duration of the catheterization for the 85 patients without endocardial lesions was 4.6 + 3.7 days (range 0.25 to 22 days).[14] Septic endocarditis also may result from S-G catheters.[15]

Therefore, the S-G catheter should not be in the pulmonary artery for any longer than four days at the maximum with an optimum of two days, unless otherwise indicated by the disease state of the patient.

Ventilator Disconnect

Surgical draping required for aseptic insertion of an S-G catheter has been associated with unrecognized ventilator disconnection, and subsequent cardiac arrest.

Surgical draping must not preclude adequate monitoring of a ventilator-dependent patient by nurses and physicians involved in the procedure.

Wedging

In order to obtain a reliable wedge pressure, the pressure transducer must be adequately calibrated and zeroed, and certain criteria must be met to insure proper catheter placement. Pressure tracings obtained during the passage of the catheter must be consistent with expected values for those heart chambers and vessels. The location of the catheter tip must be verified by chest x-rays. With a Sorensson intraflow providing continuous flushing of the S-G catheter, a stable reading of the pulmonary capillary wedge pressure must result after inflation of the balloon. If the wedge pressure is observed to increase from one oscilloscope sweep to the next, occlusion of the distal lumen must be suspected. Such a reading is not representative of the PCW pressure. The balloon should be deflated and slowly reinflated in increments of 0.2 cc until the pulmonary capillary wedge tracing is observed.

The pulmonary artery diastolic pressure is either equal to or greater than the pulmonary capillary wedge pressure. Therefore, if the PCW is greater than the PA diastolic pressure, occlusion of the distal lumen must be suspected. With the catheter in a wedged position, blood drawn for blood gas analysis should result in oxygen tensions significantly higher than the arterial oxygen tension, and the carbon dioxide tension should be significantly lower than the arterial carbon dioxide tension. This, however, is only true if the catheter tip is in a radiographically normal region of the lung. When the PA catheter is wedged in a nonaerated area of the lung, blood gases with approximate mixed venous values will result. Therefore, this criterion can be misinterpreted unless the radiographic position of the catheter tip is fully appreciated.[16]

Data From the Pulmonary Artery Catheter

In addition to the pulmonary artery pressures, blood may be drawn from the pulmonary artery catheter (with the balloon deflated) and analyzed to obtain the mixed venous oxygen tension ($P\bar{v}O_2$). The mixed venous oxygen tension represents the amount of oxygen left in the blood after the required amount of oxygen was taken up by the various organs and tissues of the body. The difference between a mixed venous oxygen content ($C\bar{v}O_2$) and the arterial oxygen content (CaO_2) is representative of the amount of oxygen removed from every 100 cc of blood exposed to the tissues in each minute [$C(a\text{-}\bar{v})O_2$].

$C\bar{v}O_2 = (Hgb \times 1.39 \times S\bar{v}O_2) + (P\bar{v}O_2 \times .0031)$

$CaO_2 = (Hgb \times 1.39 \times SaO_2) + (PaO_2 \times .0031)$

$CaO_2 - C\bar{v}O_2$ = the arterial mixed venous oxygen content differences.

$= C(a\text{-}\bar{v})O_2 = a\text{-}\bar{v}O_2$ content difference.

The $a\text{-}\bar{v}O_2$ content difference is reciprocally related to the cardiac output *(Table II)*, according to the Fick equation:

$$Qt = \dot{V}O_2 \div C(a\text{-}\bar{v})O_2$$

Qt = total cardiac output

$\dot{V}O_2$ = oxygen consumption

In order to prevent contamination of the mixed venous blood with alveolar capillary blood, the

blood samples taken from the pulmonary artery with the balloon deflated must be drawn very slowly — at a rate of no more than 1 cc/20 seconds.[16] Blood drawn from catheters which are placed too far peripherally in the pulmonary artery (in segmental or subsegmental arteries) may have higher oxygen tensions. If the oxygen content is higher than the expected mixed venous level, it probably is due to a mixture of capillary blood or diffusion of alveolar gas through the wall of the smaller vessel. Thus, blood samples drawn from a peripherally placed S-G catheter may lead to significantly large misestimates of cardiac output by the Fick method, as well as errors in shunt calculations.

The mixed venous blood is also used to calculate intrapulmonary shunt, *i.e.*, that percentage of the total cardiac output ($\dot{Q}t$) which profuses nonventilated alveoli ($\dot{Q}s$).

$$\frac{\dot{Q}s}{\dot{Q}t} = \frac{CcO_2 - CaO_2}{CcO_2 - C\bar{v}O_2}$$

where:

$CaO_2 = (Hgb \times 1.39 \times SaO_2) \times (PaO_2 + 0.0031)$
$C\bar{v}O_2 = (Hgb \times 1.39 \times S\bar{v}O_2) + (P\bar{v}O_2 + 0.0031)$
$CcO_2 = (Hgb \times 1.39 \times ScO_2) + (P_AO_2 \times 0.0031)$

Interpretation

Knowledge of the pulmonary capillary wedge pressure (a pulmonary capillary hydrostatic pressure) is of critical significance in the assessment of pulmonary congestion. It is the principal determinant of the transudation of fluid from the vascular to the extravascular space of the lung.

The plasma oncotic pressure is an osmotic pressure exerted by the plasma proteins that brings about a retention of fluid within the capillaries. Plasma oncotic pressure is in equilibrium with the opposing forces (capillary blood pressure + tissue oncotic pressure + surface tension pressure) that tends to promote pulmonary edema. Capillary blood pressure is a hydrostatic force that tends to push fluid from the vascular to the extravascular space of the lung. The tissue oncotic pressure and the surface tension of the alveoli tend to pull fluid from the vascular to the extravascular space of the lung. When these various forces are in equilibrium, there is no transudation of fluid into the extravascular pulmonary space. Plasma oncotic pressure $\rightleftharpoons$ capillary B.P. + tissue oncotic pressure + surface tension pressure ($37 \rightleftharpoons 15 + 18 + 4$).[17]

However, if disequilibrium exists, either by lowering of the plasma osmotic pressure or raising the pulmonary capillary blood pressure or the surface tension of the alveoli by absence of surfactant, then transudation of fluid into the extravascular pulmonary space may occur.[17]

$$37 < 15 + 18 + (4\text{-}30)$$

Under these circumstances, diuretics should be used until the mean pulmonary artery wedge pressure is less than 18 torr, and preferably at approximately 14 torr. While the physical examination of the chest and serial x-rays can be helpful in defining the degree of pulmonary congestion, these clinical findings may follow by several hours the actual and fundamental alterations in circulatory dynamics which are accurately reflected by the pulmonary capillary wedge pressures. Thus, in a rapidly changing state, knowledge of the mean pulmonary capillary wedge pressure is invaluable. Under these circumstances, PCW pressure monitoring may obviate overtreatment with diuretics. In circumstances such as these, the level of changes in the central venous pressure is of no value and may be misleading.[1, 2, 5]

In patients with a low output syndrome (shock), knowledge of the PCW as a reflection of the left ventricular filling pressure is of practical importance. Starling's Law of the Heart indicates that more powerful myocardial contraction results from a given increase in filling pressure. It has been found that optimal left ventricular performance can be achieved in the presence of a mean pulmonary artery wedge pressure of 15 ± 2 torr.[18] If the PCW is less than 14 and the $a\text{-}\bar{v}O_2$ content difference is 4.0-5.5 ml/100 ml, and the blood pressure and pulse are within normal limits, then left ventricular function is adequate. However, if the pulmonary capillary wedge is 14 or less, with an increased $a\text{-}\bar{v}O_2$ content difference, and pulse rate and a decreased blood pressure, then an improved cardiac performance can be expected by increasing the PCW pressure within the optimal range of 15 ± 2 torr. Such dogmatic statements cannot be said of the central venous pressure. Thus, by looking at the $a\text{-}\bar{v}O_2$ content differences, as a reflection of cardiac output, and by looking at the pulmonary capillary wedge pressures, as a function of myocardial fiber length or left ventricular filling pressures, the ventricular function curves as described by Starling can be appreciated for any given patient *(Table II)*.

If the PCW is 0-4 torr, increased fluid therapy is of definite value. If the PCW is 9-12 torr and the patient demonstrates a shock syndrome with a wide $a\text{-}\bar{v}O_2$ content difference, fluid therapy sufficient to raise the PCW to 15 ± 2 torr should increase the cardiac index (C.I. = cardiac output/surface area in square meters). Pulmonary congestion may result as the PCW exceeds 18 torr, which in turn results in in-

TABLE II

Condition	*PCW torr*	*C(a-$\bar{v}$)O_2 ml/100 ml*	*C.I. L/min/m^2*
Normal	4-12	4-5.5	2.5-3.5
High output	10-18	<4	>3.5
Pulmonary congestion	18	4-5.5	<2.5
CHF	>18	>5.5	<2.5
Hypovolemia	<12	>5.5	<2.5

creased intrapulmonary shunting, progressive hypoxia, increased respiratory and cardiac work and, ultimately, in progressive congestive heart failure.

Data from the Swan-Ganz catheter has afforded a more precise monitor of the optimal level of PEEP required to ameliorate oxygenation failure. "Optimal" PEEP related only the PaO_2 to a "good" level of PEEP, while "best" PEEP related compliance to a "good" level of PEEP.[18, 19] Both optimal and best PEEP failed to recognize the need to accurately assess the effect of PEEP on the cardiovascular (C-V) system. No attempts were made in those studies[19, 20] to augment the C-V system. However, Civetta, Kirby, and Downs[21, 22] in essence stated that we should take the opportunity afforded us by S-G monitoring to accurately assess the C-V system *(Table III)*, thereby providing the level of PEEP which results in the best lung function (Opportunistic PEEP).

PEEP should then be increased to afford a $\dot{Q}s/\dot{Q}t$ less than 0.15 (PaO_2 greater than 65 torr at FIO_2 less than 0.4) while sustaining cardiac output by appropriate evaluation and augmentation of the cardiovascular system.

The cardiovascular system is evaluated by accurate measurement of the heart rate, mean arterial pressure (MAP), CVP, mean pulmonary artery ($\overline{PA}$), pulmonary capillary wedge pressure (PCW), cardiac output (CO), and height and weight. This measured data is then used to obtain derived data, which is more useful in the evaluation of the pathophysiologic state of the C-V system and its response to therapy.

Summary

The Swan-Ganz catheter has afforded more accurate assessment of the cardiovascular system. However, attention must be paid to avoiding the incumbent complications of pulmonary infarction, perforation of the pulmonary artery, arrhythmias, infection, thrombophlebitis, knotting, aseptic endocarditis, and ventilator disconnect. The data derived from the S-G catheter requires proper sampling of mixed venous blood for blood gas analysis, calculation of derived data (C(a-$\bar{v}$)O_2, $\dot{Q}s/\dot{Q}t$, SV, CI, SVR, LVSW, etc.), and proper techniques in analog wave analysis for accurate assessment of the PCW (left ventricular filling) pressure.

TABLE III

OPPORTUNISTIC PEEP

Provide PEEP sufficient to decrease $\dot{Q}s/\dot{Q}t \leq 15\%$ while evaluating and supporting the cardiovascular system.

Preload	*Normal*
PCW	4-12 torr
CVP	2-10 torr
Contractility	
CO —	5 L/min
CI = CO ÷ BSA	2.5-3.5 L/min/M^2
SV = CO ÷ pulse	60-70 ml/min
SI = SV ÷ BSA	35-45 ml/min/M^2
LVSW = SL× MAP × 0.0136	51-61 gm-M/M^2
RVSW = SI × PA × 0.0136	8-12 gm-M/M^2
Afterload	
MAP = Diastolic + (pulse pressure ÷ 3)	85-95 torr
SVR = [(MAP-CVP)CO] × 80	900-1200 dynes sec · cm^{-5}
PVR = [($\overline{PA}$-PCW)CO] × 80	150-250 dynes sec · cm^{-5}

(Continued on page 529)

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Analgesic Agents

A Spectrum of Pediatric Criteria

ANTHONY E. FRANCIS, M.D.* *and* MEDO MIRZA, M.D.,† *Wichita*

PAIN as a *physical* event occurs when pain fibers are stimulated at a locus of injury. These fibers travel to the central nervous system (CNS). Here the message they carry is modified. The resultant modification can be called the *experience* of pain. This concept is illustrated in *Figure 1*. It has been observed for many years that the experience of pain cannot be related solely to the number of pain fibers stimulated as the result of an injury.[1] The mood of the central nervous system (anxiety, fear or euphoria, for example) is extremely important. We have all seen anxious children scream in apparent agony at the mere touch of the examiner's hand or stethoscope. This represents an extreme example in which anxiety and fear have turned an innocuous event into a truly painful experience for the child. On the other hand, there are calm and trustful children who tolerate relatively painful procedures well. The wise clinician learns to gain the patient's trust whenever possible. It is an excellent tool for reducing anxiety, and its usefulness cannot be overemphasized.

Analgesic Agents

Very few analgesic drugs are widely accepted for use in children. Among non-narcotic agents (aspirin and related compounds), four are utilized clinically:[2] aspirin (acetyl salicylic acid); salicylic acid; sodium salicylate; and methyl salicylate. Aspirin is the only drug in this category approved for use in children. This drug is orally effective; it may also be used rectally but with less predictable absorption. The action of this drug is thought to be central in origin. *Figure 2* shows the absorption and major degradation pathways of aspirin. It is passively absorbed in the unionized form from the stomach and upper small bowel. As aspirin is a weak acid, an increase in gastric acidity (decrease in pH) will increase the rate of absorption. Appreciable plasma concentrations are usually attained 30 minutes after oral administration. It is distributed to most body fluids. From 50-90 per cent of aspirin is bound to plasma proteins. Most of the free aspirin is quickly converted by plasma esterases to salicylic acid. Both aspirin and salicylic acid are metabolized in the microsomal and mitochondrial fractions of the liver to various metabolic products. These metabolic products are water soluble and are excreted in the urine. Ten to 85 per cent of salicylic acid may be excreted unchanged in the urine. An alkaline urine encourages the excretion of unmetabolized salicylic acid.

Some confusion exists in the medical world concerning the use of "adult" analgesic agents in children. Only a very few of these agents are widely accepted in pediatrics. The pharmacology, antipyretic activity, anti-inflammatory activity, dosage schedules, and poisonings with these agents are reviewed.

Side effects of aspirin therapy include gastritis, increased respiration, and allergy.[3] Bleeding tendency may occur. This is related to inhibition of prostaglandin synthetase activity on the surface of the platelets. People who have pseudocholinesterase activity in their plasma have been reported to have increased metabolism of aspirin to salicylic acid.[4] Patients with Criggler-Najjar syndrome as well as their parents and siblings may have problems conjugating salicylates.[5] A family history should always

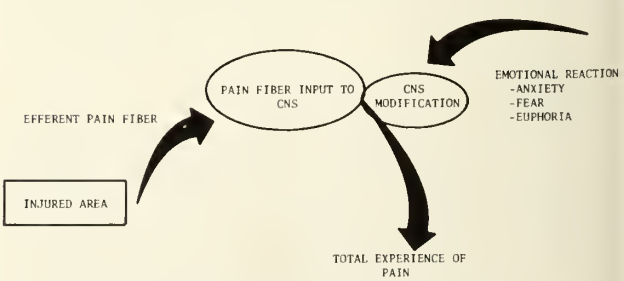


Figure 1. Total perspective of pain.

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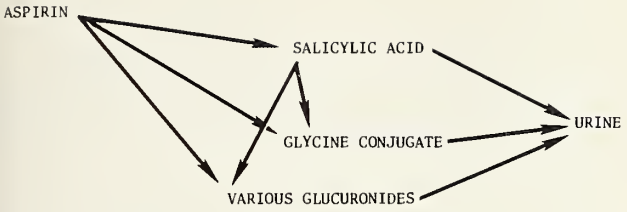


Figure 2. Degradation pathways of aspirin. The degradation of aspirin to salicylic acid occurs mainly in the plasma by esterases. Conjugation with glycine and glucuronides occurs in many tissues, but mostly in the liver microsomal and mitochondrial fractions.[22]

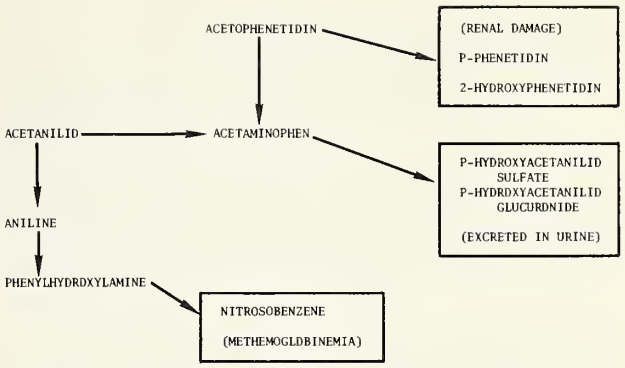


Figure 3. Degradation pathways of para-amino phenols. The metabolic products of acetophenetidin (phenacetin) have been implicated as causative agents in chronic renal damage. The degradation pathway of acetanilid yields nitrosobenzene, a cause of methemoglobinemia.[8]

be checked for glucose-6-phosphodiesterase deficiency (G-6-PD) before any analgesic, especially aspirin, is prescribed.[6] Aspirin has been reported to cause hypoglycemia that is not related to increased insulin levels.[7] This is a rare effect.

The para-amino phenols group[2, 8] consists of three analgesic agents — acetaminophen, acetanilid, and acetophenetidin (Phenacetin) — of which only acetaminophen is used in pediatrics. It can be seen from *Figure 3* that acetanilid and acetophenetidin are metabolized in the body to acetaminophen. This occurs in the microsomal system of the liver. Acetanilid is also converted to aniline. The metabolic products of aniline, phenylhydroxyamine, and nitrosobenzene are the agents associated with methemoglobinemia production in the body. For this reason, acetanilid is no longer recommended for clinical use.[2]

Acetaminophen gives good analgesia for mild to moderate muscle and joint pain and headache. It is usually ineffective for intense pain, such as occurs with smooth muscle spasm. The drug when administered orally is effective and is rapidly absorbed from the gastrointestinal tract. Peak plasma levels usually occur in 30-60 min. Variable binding to plasma proteins occurs. Adverse effects include hepatic and renal tubular necrosis. This is the product of acute poisonings. As with any drug, an allergic reaction may occur.

Narcotic agents[3, 9] include morphine sulfate, meperidine HCl, codeine phosphate, methadone, and paregoric. The mechanism of action of this class of drugs remains obscure. They do not affect peripheral pain receptors to any great extent. They do apparently affect the CNS modification of pain, so that the painful stimulus is no longer perceived as detrimental. In addition to the analgesic effect, drowsiness, mood changes, and mental clouding may occur. Dysphoria with an increase in anxiety may also be produced, especially in children. Morphine sulfate and meperidine HCl are effective against dull or sharp pain. Dosages may have to be increased for sharp pain analgesia. Codeine phosphate is usually used for mild to moderate pain.

Many physiological effects are observed with the narcotic agents. Pupillary constriction occurs via the Edinger-Westphal nucleus. Respiratory depression is the result of brain stem depression. Nausea and resultant vomiting are mediated through stimulation of the chemoreceptor trigger zone. Peripheral vasodilatation is thought to be due to the release of histamine caused by the narcotic. A decrease in smooth muscle activity is noted throughout the body. This is the partial mechanism for analgesia in patients suffering kidney stones or other diseases related to smooth muscle spasm. A general decrease in GI motility is seen. Along with this is a decrease in almost all secretory activity of the GI tract, including stomach, pancreas, and biliary tract. Respiratory depression with a retention of CO_2 may lead to cerebral vasodilation and a resultant increase in CSF pressure. Meperidine HCl has been specifically cited by one reference[10] as offensive in this regard. For this reason, narcotics should be avoided in states such as hydrocephalus. Morphine and meperidine HCl may produce bronchospasm; however, this is rarely seen after therapeutic doses. Theoretically, narcotics may cause problems with asthmatics.

The opioids are readily absorbed from the GI tract. There may be a decrease in therapeutic effectiveness if administered orally. These drugs are also active when given intravenously, intramuscularly, subcutaneously, or by inhalation.

As with any drug, narcotics can trigger allergic reactions.

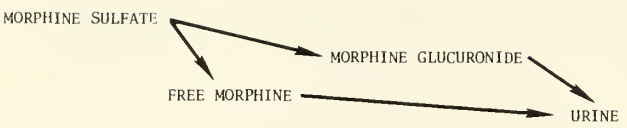


Figure 4. Simplified degradation pathways of morphine sulfate. Degradation takes place in the microsomal system of the liver.[22]

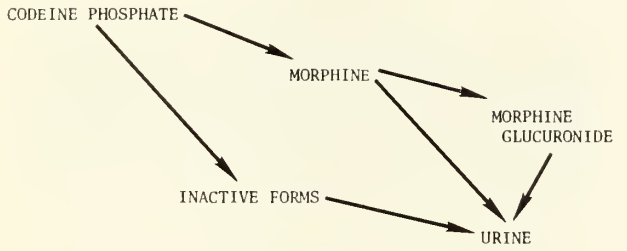


Figure 5. Degradation pathways of codeine phosphate. Codeine may be degraded to form morphine. This occurs by demethylation. Degradation of morphine then follows the same pathways as described in *Figure 4*. Codeine is also degraded in the liver to various inactive forms which are excreted in the urine.[22]

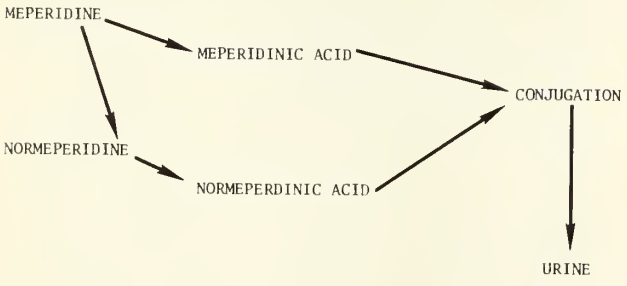


Figure 6. Degradation of meperidine HCl. Meperidine is hydrolyzed to meperidinic acid. It is also N-demethylated to normeperidine which is hydrolyzed to normeperidinic acid. These products are then conjugated with various compounds and excreted in the urine. This pathway takes place in the liver.[22]

Antipyretic Action

Aspirin and acetaminophen are the analgesic agents commonly used in pediatrics that also exhibit antipyretic action.[11] Sodium salicylate also has antipyretic action. In an acute febrile episode, aspirin and acetaminophen have about the same efficacy. Sodium salicylate is about 50 per cent less potent than aspirin as an antipyretic. It may also be more irritating to the gastric mucosa than aspirin. For this purpose, the following dosages are recommended:

Aspirin (and other salicylates)	10-20 mg/kg/6 hrs
Acetaminophen	5-10 mg/kg/6 hrs

The salicylates are the only analgesic agents that have any anti-inflammatory action.

Analgesic Dosage[2, 10, 12]

Acetaminophen given orally	under one year 60 mg 1-3 years — 60-120 mg 3-6 year — 120 mg 6-12 year — 240 mg may repeat every 4-6 hours or 0.7 gm/meter^2/24 hrs in 4-6 equal doses
Aspirin given orally or rectally	65 mg/kg/24 hrs (maximum 3.6 gm in 24 hours) or 1.5 gm/meter^2/24 hrs in 4-6 equal doses

Note: Good results are obtained with either Aspirin or Acetaminophen given one grain (60 mg) for each year of life up to six years in 4-6 equal doses. This is easy to remember.

Codeine phosphate*	3 mg/kg/24 hrs or 100 mg/meter^2/24 hrs in 6 equal doses
Meperidine HCl*	6 mg/kg/24 hr (maximum 100 mg/dose) or 175 mg/M^2/24 hr in 6 equal doses
Morphine sulfate†	0.1-0.2 mg/kg/dose (maximum 15 mg/dose)
Methadone HCl*	0.7 mg/kg/24 hr or 20 mg/M^2/24 hr in 4-6 equal doses
Paregoric (contains 0.4 mg morphine/ml) given orally	0.25-0.5 ml/kg dose

Poisonings with Analgesics

Aspirin: Salicylate poisoning is believed to be the most common poisoning in children, with younger children the most commonly affected. It is manifested by vomiting, tinnitis, hyperpnea, fever, lethargy, and mental confusion. Dehydration is likely to be present by the time the child is brought to medical attention.[13-15]

Older children and adults tend to suffer respiratory alkalosis, while young children and infants tend to drift into metabolic acidosis in poisoned states. Hypoglycemia or hyperglycemia may be present. Serum salicylate levels should be determined. In cases of chronic abuse, these levels may be mis-

* May be given orally or by injection.
† Subcutaneous injection.

leading. Blood pH and gases should be drawn to determine the acid-base status.

THERAPY: If the child is seen within 4-6 hrs of the ingestion, syrup of Ipecac and activated charcoal may be given. If syrup of Ipecac is ineffective in initiating vomiting, apomorphine may be administered subcutaneously. Vomiting should never be attempted if the child is unconscious. Apomorphine toxicity may be reversed with a narcotic antagonist. A stomach pump may be needed if all else fails.

Parenteral fluid support should be provided along with appropriate correction of any gross abnormalities in blood pH. Sponging may be necessary for hyperpyrexia. External respiratory support may be needed in severe cases. As a last resort for persistent cases that fail to respond to more conservative therapy, extracorporeal dialysis, peritoneal dialysis, or exchange transfusion could be used.

Acetaminophen: Poisoning with this drug is made evident by nausea, vomiting, anorexia, and abdominal pain. In adults, as little as 10-15 gm has caused hepatotoxicity. A dose of 200-250 mg/kg (about 25 gm in adults) is potentially fatal.[2, 12] In acute or chronic poisonings, renal tubular necrosis as well as increases in SGOT and LDH can be seen.

THERAPY: The therapy is essentially the same as in aspirin poisoning. Induced emesis, gastric lavage, and external support with parenteral fluids and assisted ventilation should be initiated. Methionine and cysteamine given intravenously have been reported to prevent, or make less severe, hepatic necrosis which can occur in acute poisonings.[16, 17] Hemodialysis or exchange transfusion may be used as needed. These last measures may not reverse the effects of hepatotoxicity.

Narcotics: Poisoning with narcotic agents may cause an initial excitatory stage followed by depression of all neurological functions of the CNS. This may lead to somnolence, coma, and respiratory depression.[9, 18, 21]

THERAPY: Establishment of an airway should have first priority as respiratory depression is a likely event in this poisoning. Gastric lavage may be carried out with activated charcoal, potassium permanganate, or tannic acid.

Three narcotic antagonists are commonly used in the clinical setting. These are nalorphine (Nalline — 0.100 mg/kg), levallorphan (Lorfan — 0.020 mg/kg), and naloxone (Narcan — 0.005 mg/kg). Each of these drugs is effective. It appears that only Narcan will reverse the effects of pentozocine poisoning, but it has not been officially approved for use in children.

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Oxygen Toxicity

Oxygen–Dr. Jekyl or Mr. Hyde?

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During the past decade, the widespread therapeutic use of oxygen has brought the problem of pulmonary oxygen toxicity to the awareness of clinicians. The literature on oxygen toxicity is briefly reviewed and guidelines for safe utilization of oxygen are presented.

Toxic effects of high oxygen tensions upon the lungs have been widely recognized as an important clinical problem only during the last decade. Ironically, the problem has arisen out of improvements in techniques of caring for patients with hypoxemia. Methods for administration of high inspired oxygen concentration for long periods of time are available in even the most remote medical care facilities in the United States. Other respiratory care techniques, most notably mechanical ventilation, have made it possible to prolong life in many patients with hypoxemic respiratory failure who would previously have died before pulmonary oxygen toxicity could become manifest. It is now common for patients to remain alive for many weeks with the assistance of mechanical ventilation and high inspired oxygen concentrations. The threat of oxygen toxicity in such patients is very disturbing, and questions frequently arise concerning safe levels of inspired oxygen and duration of treatment in such patients. As will become apparent from the discussion below, these questions often cannot be answered precisely from data currently available. A sensible pragmatic approach to patient care can be formulated however, and is summarized below.

Biologic Process

It is fascinating to consider that the potential toxicity of oxygen, only so recently emphasized in general medical literature, was predicted in 1775 by one of the discoverers of this gas. A recent review by Winter[1] quotes Priestly, who anticipated that human life might be expended quickly, as a candle flame burns out, in a pure oxygen atmosphere. "A moralist at least, may say that the air which nature has provided for us is as good as we deserve."

Since the discovery of oxygen, a vast store of knowledge has been accumulated concerning the effects of the element on biologic processes. This literature has been exhaustively reviewed recently,[1-6] and it is clear that our knowledge is most incomplete where it concerns medical personnel the most. The effects of exposure of normal and diseased humans to varying oxygen concentrations has been relatively little studied. Experiments on animals, isolated organs, and tissue cultures are, for obvious reasons, much more readily carried out, and much information is available. Great variation has been found in the susceptibility of various tissues and species to the effects of oxygen. This observation in itself limits the usefulness of attempts to apply to man data obtained in other animal species.

Regardless of this limitation, however, certain generalizations can be drawn from non-human studies which are of help in understanding the problem of pulmonary oxygen toxicity in man. First, it is clear that regulation of the rate of oxidation-reduction reactions is essential to the function of all biologic organisms. Therefore, all species and cells appear to be subject, to a greater or lesser degree, to the influence of variation in oxygen tension. This influence may be exerted by alterations in concentration or barometric pressure. In general, the influence is a graded one over a wide range of oxygen tensions, rather than one with a threshold. The influence may be recognized only biochemically, and may be reversible; or it may be sufficiently severe to produce structural changes and be irreversible; it may or may not alter function of the organism enough to be recognized as "toxic." From these observations, one would expect pulmonary oxygen toxicity in man to occur over a range of oxygen tensions produced by changes in concentration or in barometric pressure; and to be manifest with a range of severity and reversibility.

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Variations in Resistance

The first observations of oxygen toxicity were made during the late 19th century in the course of hyperbaric studies carried out in high pressure chambers.[1-4] Some of these observations concern oxygen tensions much higher than the 760 mm Hg maximum that can be achieved with pure oxygen under "isobaric" therapeutic conditions (at sea level barometric pressure). Therefore, the first recognized toxic effect of oxygen was an acute central nervous system (CNS) manifestation in the form of grand mal seizures. Acute CNS oxygen toxicity occurs with brief exposure to very high oxygen tensions (minutes to hours at 2.5 atmospheres of pure oxygen, or the equivalent).

It was somewhat later recognized that relatively lower oxygen tensions (50 to 100 per cent oxygen at sea level barometric pressure) can be tolerated for long periods of time without production of acute CNS toxicity, and will produce pulmonary toxic changes over a period of hours to days.

Damage to other organs by high oxygen concentrations is limited by the organism's ability to survive CNS and lung damage. Damage to the eye in immature animals, to testicular tissue, and to erythrocytes does occur, however. Variation in organ and tissue sensitivity to oxygen toxicity is due to differences in blood supply and other factors influencing local tissue oxygen tension, as well as individual cellular differences in sensitivity.

Marked differences in sensitivity to oxygen toxicity have been observed among animal species, with cold-blooded animals appearing most resistant.[5] Individual variation is also considerable. Most mammalian species with the exception of primates exhibit a survival time at one atmosphere of pure oxygen in the range of 50-100 hrs. Primates are relatively resistant, surviving in the neighborhood of 400 hrs. Among the cold-blooded species, turtles have been reported alive and unharmed at 240-552 hrs, while frogs may survive for more than 1,000 hrs. These data clearly indicate caution in applying animal studies of oxygen toxicity to man.

Experimental work in animals has, however, identified certain factors that influence sensitivity to oxygen toxicity, and that may provide a basis for the development of prophylactic and therapeutic measures in the future.[5] Acquired resistance in survivors of oxygen toxicity may be related in part to arterial hypoxemia from structural lung damage; increased capacity of lymphatics to clear edema fluid; mitochondrial changes; a modified adrenocortical or adrenomedullary response to stress;[5] and an increased capacity of normal antioxidant defense mechanisms.[7, 8] Increased resistance to oxygen toxicity has been observed in immature animals, as well as in those subjected to fasting and hypothermia and those treated with antioxidants such as Vitamin E. Greater sensitivity to oxygen toxicity has been induced by hyperthermia, administration of adrenal and thyroid hormones, deficiency of Vitamin E, and roentgen irradiation.

Animal Studies

As has been pointed out above, there is a great deal of species variation in sensitivity to oxygen toxicity. In addition, the pathologic changes seen in the lungs of animals that have died from oxygen toxicity vary with the duration and intensity of the exposure that produced death. In particular, brief exposure to very high oxygen tensions may result in early death due to CNS toxicity with relatively little lung damage. Some generalizations can be made, however.

Studies in animals exposed to about 1.0 atmosphere of oxygen (100 per cent oxygen at sea level barometric pressure) provide information about the course of oxygen toxicity that might occur under usual therapeutic conditions.[5] Under such conditions, rats, mice, and rabbits show changes of pulmonary congestion and edema after two-three days. Hyaline membrane formation then occurs, with destruction of capillary endothelial cells and hemorrhage from damaged capillaries. Proliferation of alveolar cells and fibrosis follows. Some animals succumb rapidly, after only three or four days of exposure, while others survive ten days or longer.

The acute changes of pulmonary edema appear to be completely reversible in animals removed from high oxygen exposure; even hyaline membranes appear to regress. Proliferative changes occurring from prolonged exposure produce irreversible scarring in animals that survive.

The mechanisms by which high inspired oxygen tensions cause damage to the lung appear to be multiple and complex.[1-5] Without doubt, direct toxicity to cells of the lung occurs by inactivation of sulfhydryl enzymes, perhaps through formation of free radicals. The biochemistry of direct oxygen toxicity to cells has recently been reviewed in detail.[4] This direct cytotoxic effect may be mediated by contact of alveolar gas itself with alveolar cells, as well as by high PO_2 of pulmonary capillary blood and arterial blood perfusing the bronchial circulation. In addition, however, indirect lung damage mediated by the central nervous system and adrenal cortex and medulla has been convincingly documented, although it is not well understood. These effects presumably depend on high PO_2 of arterial blood. Fi-

nally, absorption atelectasis contributes to lung damage, as high oxygen concentrations are ordinarily achieved by displacement of inert gas (nitrogen) by oxygen. Decreased surfactant probably plays a role in atelectasis as well.

The multiplicity of the mechanisms of pulmonary oxygen toxicity helps to explain the variability in results of experimental studies done under varying conditions. One of the most important questions that has been studied by animal experimentation, with inconclusive results, concerns the possibility that pulmonary oxygen toxicity may be modified by prior lung damage.[9-11] It seems entirely possible that those toxic effects mediated by the CNS and by endocrine glands might be lessened if the lungs are unable to transmit high oxygen tensions to the arterial blood perfusing these structures. In addition, the oxygen tension of blood perfusing the bronchial circulation might influence oxygen toxicity; and the possibility exists that structural damage to the lung may somehow alter the sensitivity of cells to direct toxic effects of oxygen. If such is the case, then most patients requiring high levels of inspired oxygen in therapy might have protection against toxicity by virtue of their disease.

Animal studies addressing themselves to this problem have demonstrated that anatomic right to left shunting is protective against some of the pulmonary change caused by hyperbaric oxygen toxicity associated with convulsions.[9] However, shunting does not appear to protect the lungs against oxygen toxicity produced by less intense exposure—that is, isobaric exposure without convulsions.[10] One recent study[11] suggests that lung damage produced by oleic acid injection provides some protection against oxygen toxicity at isobaric levels. To the extent that this experimental model reproduces lung disease in humans, it is somewhat reassuring to respiratory care personnel who often must use high levels of inspired oxygen to sustain life in patients.

Toxicity in Man

Certain difficulties are inherent in studies of oxygen toxicity in man. Experiments in normal volunteers can be carried out for only brief periods, until symptoms and physiologic abnormalities can be first observed, but are reversible. No pathologic data can be obtained from such experiments. Patients who die of oxygen toxicity are a source of pathologic information; but in most cases these data are enormously confused by severe lung damage from other disease.

Symptoms of oxygen toxicity in normal volunteers breathing 100 per cent oxygen at one atmosphere begin at about six hours. Cough and substernal pain suggest tracheitis initially; a recent study has documented the appearance of tracheitis and slowing of tracheal mucus velocity by bronchoscopic techniques,[12] followed by dyspnea, paresthesias, nausea and vomiting, and fatigue. Voluntary tolerance usually is ended at between 50 and 75 hrs of exposure, with a maximum of 110 hrs reported. Physiologic abnormalities of oxygen toxicity documented by studies in normal volunteers have included decreased lung compliance, reduced vital capacity and total lung capacity, and impaired diffusing capacity.[5]

Lower intensity exposure can be tolerated for longer periods of time. Fifty-five per cent oxygen at one atmosphere has been tolerated for seven days, although with substernal discomfort beginning on the second day. Thirty-five per cent oxygen has been tolerated without ill effects for 30 days.[5]

Widespread clinical interest in the problem of oxygen toxicity from therapy in man was generated by a group of retrospective studies in 1967. Nash and associates[13] presented morphologic data on 70 patients who died after prolonged mechanical ventilation. Pathologic changes were observed to be characteristic, and resembled those seen in experimental oxygen toxicity. In addition, the severity of the changes correlated with the level of inspired oxygen which had been used, but not with duration of mechanical ventilation. This study was not purported to demonstrate a clear cause-and-effect relationship between oxygen therapy and lung damage, but it was reasonably suggested by the authors that higher than necessary oxygen concentrations should be avoided in therapy.

The report of Northway[14] reviewed data from 32 newborn infants with respiratory distress syndrome who had been treated with high oxygen concentrations and mechanical ventilation. Again, pathologic findings in those infants who died bore some resemblance to experimental oxygen toxicity. The possibility could not be excluded that the changes were a late manifestation of the primary disease process in subjects who would not have survived as long without high oxygen and ventilator support.

Shanklin and Wolfson[15] studied retrospectively a group of newborns who died and were autopsied. Eighty-three cases were studied in which data available were sufficient for detailed evaluation. They were grouped according to duration of oxygen administration, and significant correlation between oxygen therapy and pulmonary hemorrhage was found.

The major difficulty in interpreting these studies is

the one that still plagues the individual physician who tries to evaluate pathologic information on one of his patients who fails to survive long-term ventilatory support. Oxygen toxicity cannot be identified so well histologically as to be clearly separated from the damage produced by primary lung diseases of various sorts.[16] Neither the clinical course nor the histology of oxygen toxicity are entirely distinctive, although they may be characteristic. Each of the pathologic changes described in oxygen toxicity is a rather nonspecific response that can be produced by a number of insults.

In addition, it should be recognized that prolonged mechanical ventilation carries with it the possibility of a number of potentially damaging events other than oxygen toxicity. Small, uniform tidal volumes; removal of the protective cough mechanism; and monotonous positioning of the patient are only a few of the potentially damaging factors that may accompany mechanical ventilation and produce pulmonary injury that may be identified as oxygen toxicity.

In response to the questions raised by these retrospective studies, three important prospective studies were undertaken and were subsequently published in 1970.[17-19] Along with data from animal and other human studies, these investigations provide adequate guidelines for the safe therapeutic use of oxygen, even in the absence of detailed and precise dose-response information at many levels of intensity and duration.

Van DeWater and associates[17] reported a study of 11 healthy volunteers exposed to breathing 100 per cent oxygen for 6-12 hours. Parameters studied and found to be unchanged from this exposure included alveolar-arterial oxygen gradient, physiologic pulmonary shunts, pulmonary artery pressure, total pulmonary resistance, cardiac output, and pulmonary extravascular water volume. The first two parameters listed, in particular, could be expected to be sensitive and reliable indicators of early abnormalities of pulmonary oxygen toxicity. Therefore, their failure to change appreciably during the study can be viewed as strong evidence for the safety of an exposure to pure oxygen for 6-12 hrs.

Singer and colleagues[18] studied two groups of patients receiving mechanical ventilation following cardiac surgery. A group of 20 patients received the minimum inspired oxygen concentration required to maintain an arterial oxygen tension of 80-120 mm Hg. (None required more than 42 per cent oxygen.) A group of alternately selected patients received pure oxygen. After a mean exposure time of 21 hrs in the former group and 24 hrs in the latter, no difference was found in intrapulmonary shunt, effective compliance, ratio of dead space to tidal volume, or clinical course. Those abnormalities occurring in these parameters might then be attributed to pre-existing disease, surgical insult, and effects of mechanical ventilation other than high inspired oxygen tension. This study provides some evidence of the safety of an exposure to pure oxygen of 24 hrs duration.

Barber and associates[19] studied patients receiving mechanical ventilation following irreversible brain damage. Five patients received ventilation with room air and five with pure oxygen. Subsequent autopsy revealed physiologic abnormalities and pathologic changes in both groups. However, arterial oxygen tension during pure oxygen breathing fell more rapidly in the oxygen group after 30 hrs, proving to be a parameter for distinguishing between the two groups. Microscopic examination of the lungs did not distinguish between the groups, but abnormalities were extensive in both. This study suggests that untoward effects of high oxygen concentrations on the lungs may become manifest after 30 hrs of exposure. The findings are complicated by extensive abnormalities in both control and experimental groups, perhaps secondary to brain damage.

Conclusion

In spite of the enormous literature which is available on the subject of oxygen toxicity, no specific, clear guidelines can be provided as to maximum concentrations of oxygen that can be tolerated safely for various durations of time in human subjects with lung disease. Nevertheless, an entirely sensible and simple approach to this problem will avoid needless risks, and permit satisfactory patient care.

The cornerstone of this approach is the rule that inspired oxygen concentrations should never be used that are higher than needed to produce an arterial oxygen tension adequate for tissue oxygenation. Measurement of arterial blood gases must be carried out in order for this rule to be followed, and methods of measuring and controlling inspired oxygen concentration must be available. The definition of an "adequate" arterial oxygen tension is subject to interpretation, based on individual patient problems. Seldom is a PaO_2 greater than 80 mm Hg needed, as the oxygen carrying capacity of hemoglobin is nearly saturated at that tension, and further increments in oxygen tension produce very little change in oxygen content of blood. In fact, hemoglobin saturation is adequate for good tissue oxygenation at PaO_2 of 60 mm Hg, and chronically hypoxemic patients usually tolerate PaO_2 of 50 mm Hg quite well. Oxygen saturation of hemoglobin falls off rapidly below this level, however, and oxygen tensions lower than 50

mm Hg usually should not be considered acceptable. The presence or absence of cardiac arrythmias or CNS symptoms attributed to hypoxia might influence the level of PaO_2 between 50 and 80 mm Hg chosen to be maintained in an individual patient.

In general, an inspired oxygen concentration greater than 50 per cent should be avoided if possible when oxygen supplementation is expected to be needed for several days. Available data suggests that lower levels are well tolerated for days or weeks.

When pure oxygen is required, it is probably relatively innocuous if used for less than 24 hours. Considerable effort should be expended to avoid the need for its use for greater than this length of time.

Measures available to decrease the need for high inspired oxygen concentration include specific therapy directed at the underlying disease process. Fluid management, for example, is of great importance in pulmonary edema from any cause, but especially in volume overload or left ventricular failure. Adequate tracheal suction, adequate tidal volumes or intermittent "sigh," frequent position change, and use of positive end expiratory pressure in selected patients may be of great help in decreasing oxygen requirements.

Adherence to these simple guidelines will prevent such needless risks as exposure of a patient to pure oxygen, producing an arterial oxygen tension of 300 or 400 mm Hg for several days. Such risks are entirely avoidable with currently available technology at acceptable cost. The requirement of availability of measurement of arterial blood oxygen tension is apparent, as is the need for ability to measure or estimate inspired oxygen concentration. These capabilities are essential in any hospital treating patients with respiratory insufficiency.

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The Dean's Letter

(Continued from page 477)

research activities. Among our volunteer, part-time and full-time faculty, a number of projects are underway. More are in the idea-forming stages and need further refinement as well as adequate funding. William Reals, M.D., was named Assistant Dean for Research, Planning, and Development in April and has taken on the challenge of coordinating research activities, as well as exploring potential funding sources. Research development is important, not only for our accreditation, but to promote new advances in the clinical medical sciences. We look ahead with anticipation to developing a cooperative research relationship with the Wichita Area Veterans Administration Center.

In looking back over the year's activities, it may seem like a "long and winding road." Indeed, it is at times. We have a responsibility to provide quality medical education and attempt to meet the needs of the Kansans who support us. There never seems to be a shortage of good ideas . . . of worthwhile projects to initiate. There does, at times, seem to be a lack of time to do everything we would like to do, as well as a framework of limited resources we must work within. Still, I would be less than honest if I failed to say that I am pleased with the development of The University of Kansas School of Medicine-Wichita. We thank you again for your support.

Castleman's Disease

Surgical Management of the Head and Neck

JOE J. LIN, M.D.,* FERNANDO R. KIRCHNER, M.D.† *and* GEORGE J. FARHA, M.D.,‡ *Wichita*

CASTLEMAN'S DISEASE, characterized by benign lesions commonly occurring in the intrathoracic area, is rarely considered in the evaluation of head and neck tumor.[1-3] Since the original description of an intrathoracic tumor in the Case Records of the Massachusetts General Hospital by Castleman[4] in 1954, there have been more than 100 cases reported in the literature.[5-9] Most of these cases were intrathoracic lesions, and head and neck lesions were relatively uncommon. Care should be taken not to confuse this entity with malignant lymphoma, angioimmunoblastic lymphadenopathy, or thymoma. Keller *et al.*[3] separated this disease into two distinct histologic patterns — the hyaline-vascular type was characterized by small hyaline-vascular follicles and interfollicular vascular proliferation; the plasma cell type was characterized by large follicles with intervening sheets of plasma cells. The following is a report of three cases of the hyaline-vascular type encountered during our 15-year experience in the area of head and neck surgery.

Report of Cases

Case One

In February 1972, four months prior to admission, an 18-year-old male noticed a sore throat which he described as the sensation of a lump with dull pain in the right side of the pharynx. Past history was not remarkable. Physical examination revealed a smooth mass arising in the posterolateral aspect of the right oropharynx. The appearance of the right tonsil was not remarkable, but it was protruding due to an extrinsic pressure. The mass seemed to be extending from the level of the hyoid bone to the base of the skull superiorly. The functions of swallowing and breathing were noted to be normal. Auscultation of this area revealed no bruit. The preoperative diagnosis was neurofibroma. Laboratory data were not remarkable. Peripharyngeal exploration was performed using the right submandibular compartment for exposure. During the procedure, a 5-cm tan, meaty, well defined tumor was encountered and resected. The vascular supply of the tumor appeared to be from branches of the right superior thyroid artery. The incision healed without complication. To date, the patient is well and active, and there is no evidence of recurrence.

Castleman's disease manifests as a benign lesion of unknown cause. Its histologic pattern may be either hyaline-vascular type or plasma cell type, and it generally occurs in the intrathoracic area. Symptoms, in addition to palpable mass, include fever, anemia and hyperglobulinemia, all of which may be alleviated or resolved by surgery. Presented here is a review of the surgical management of three cases of Castleman's disease of the hyaline-vascular type occurring in the head and neck.

Case Two

In early 1970, a 25-year-old white female reported a three-month history of a painless, slightly movable, deep-seated right paratracheal mass. No obvious associated recent episode of malaise, cough, fever, bleeding, or other significant symptoms was noted. Phonation was within normal limits, but speech impediment was present. Past history was not remarkable except for a tonsillectomy and adenoidectomy as a child, and an allergy to penicillin. Physical examination failed to reveal any abnormality except for cervical mass on the right side. The hematologist's impression was that malignant lymphoma was present, but bone marrow aspiration did not produce positive indication. Upon excision, a 3-cm well encapsulated, rubbery, tan nodule was noted. No gross evidence of necrosis was present. Eight years after excision of the tumor, the patient is

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Figure 1



Figure 2

doing well and no recurrence is observed, although the speech impediment still exists.

Case Three

In 1971, a 31-year-old Oriental female was admitted to a hospital in the Philippines presenting a deep mass in the lateral neck on the right side. A wedge biopsy was performed and a benign diagnosis was given, but no pathologic diagnosis was made known to the patient. Past history revealed chronic intractable microcytic anemia that did not appear to respond to iron pills. The tumor appeared to grow slowly and she was admitted to the hospital for evaluation. On physical examination a large, nontender, movable, non-pulsating mass was palpated deep in the middle neck on the right side. The thyroid was not enlarged and there was no lymphadenopathy. Laboratory data on admission revealed hemoglobin, 9.4 gm/100 ml; hematocrit, 30%; MCV, 57.4 μ^3; WBC, 5100/mm^3; T3, T4, within normal limits; SMA-20, normal; EKG, normal; total protein, 6.6 gm/100 ml with gammaglobulin 1.3 gm/100 ml (IgG 1467 mg/100 ml, IgA 253 mg/100 ml, IgM 116 mg/100 ml). At surgery, a 5.5-cm tan, resilient, well encapsulated tumor was discovered beneath the sternocleidomastoid muscle at the level between hyoid bone and thyroid cartilage. It was highly adherent posteriorly and appeared to have a blood supply from branches of the superior laryngeal artery. The postoperative course was uneventful. Six months after surgery the patient was seen in the outpatient clinic and laboratory findings included hemoglobin, 11.3 gm/100 ml; hematocrit, 37.6%; MCV 67.7 μ^3. To date, there has been no recurrence of the lesion.

Pathology

Gross: Each tumor was well encapsulated with no evidence of infiltration of the surrounding structure *(Figure 1)*. The size ranged from 3.0-5.5 cm. The cut surface was homogeneously tan and was devoid of hemorrhage or necrosis *(Figure 2)*. The consistency was rubbery to meaty.

Microscopy: All three cases showed a picture of the hyaline-vascular type of Castleman's disease.[3] The tumors were all encapsulated by a rather thick capsule with no discernible subcapsular sinuses. The tumor proper was composed of multiple follicles with epithelioid cells in the centers surrounded by a concentric array of mature lymphocytes *(Figures 3, 4)*. The interfollicular areas showed proliferating capillaries, some of which had a tendency to penetrate the follicles to communicate with the epithelioid cells *(Figure 5)*. The degree of hyalinization varied from area to area and case to case. Mature lymphocytes and plasmacytoid cells were scattered in between the capillaries.

Discussion

Terminology: This lesion has been described under a variety of names,[1, 2, 5-7, 10-13] including

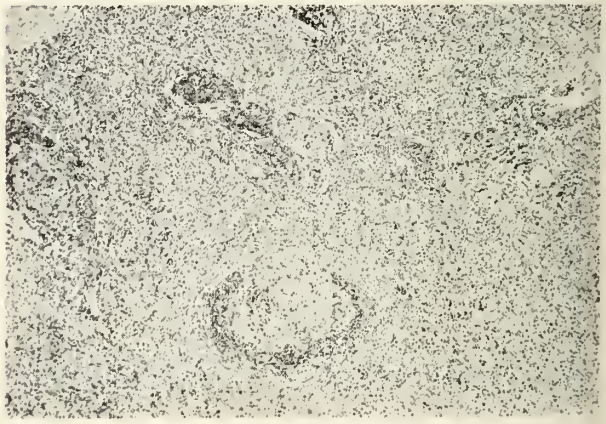


Figure 3

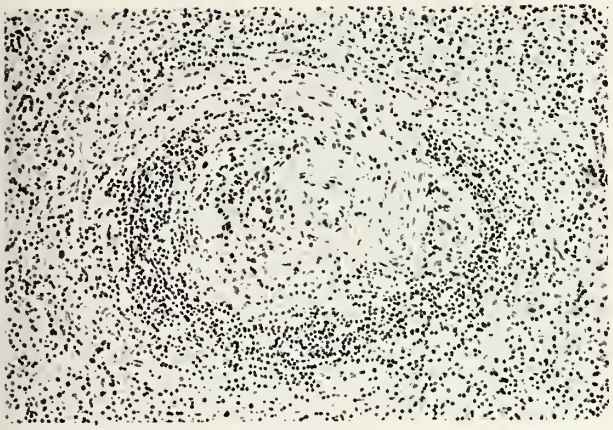


Figure 4

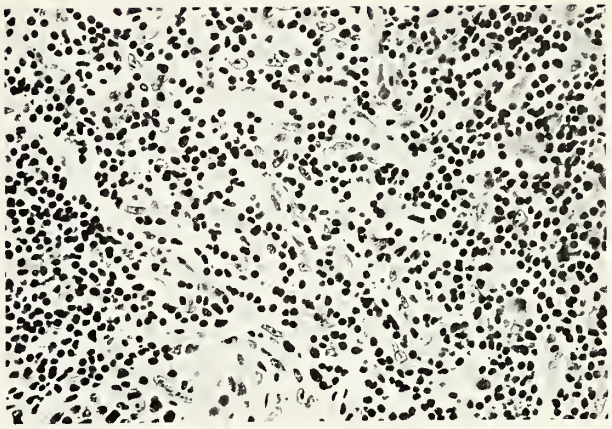


Figure 5

lymph node hyperplasia resembling thymoma, lymph nodal hamartoma, thymic choristoma, tumor-like proliferation of lymphoid tissue, giant intrathoracic lymph node, follicular lymphoreticuloma, angiofollicular mediastinal lymph node hyperplasia, angiomatous lymphoid hamartoma, benign giant lymphoma, and Castleman's disease. Such terms as lymph node hyperplasia and giant lymph node appear inappropriate as this lesion has been observed in areas where lymph nodes are not normally present (Case 1). Both benign giant lymphoma and lymphoreticuloma imply that this lesion is a neoplasm, which has not been well established. Hamartoma theory seems plausible, yet it is controversial (see *Causes*). Until the cause is known, it appears reasonable to accept the term Castleman's disease, as Castleman was the first to describe and study the lesion extensively.

Causes: Castleman's disease is a pathologic entity of unknown cause. There are two important hypotheses: (1) hamartoma; and (2) inflammatory process. Tung and McCormack[2] favor the hamartoma theory, pointing out the presence of a distinctive and large systemic arterial supply to the lesion, a vascular circulation different from a normal lymph node, active vascular proliferation, and stromal vascular sclerosis similar to other hamartomas. However, unlike congenital mesenchymal tumors[14] which are well accepted as hamartomas, no congenital cases of this lesion have been described. The presence of plasma cells and plasmacytoid lymphocytes in this lesion does not favor the hamartoma theory. Furthermore, the syndrome of fever, anemia, and hyperglobulinemia (Castleman's syndrome) associated with this lesion can hardly be related to a hamartoma.

Keller and associates[3] contended that the presence of eosinophils, plasma cells, and vascular proliferation was suggestive of inflammatory process and cited viral lymphadenitis as a similar example. However, no causative agents have been identified. The concept of choristoma,[7] also seems unlikely as the formation of a lymphoid nodule in various extranodal locations has been well documented. Chronic follicular cervicitis of uterus and chronic follicular cholecystitis are good examples. In the current three cases, we were unable to pinpoint the causative agent. The remarkable finding is that in Case 3, after removal of the lesion, the intractable chronic microcytic anemia was much improved — a finding also reported by others.[15, 16]

Differential diagnosis: The differential diagnosis in the case of Castleman's disease (hyaline vascular type) should include enlarged lymphoid lesions with follicular structures.

Nodular lymphoma may be confused with Castleman's disease because of the follicular structures throughout the entire lymphoid mass; however, the presence of hyaline vascular follicles and the absence of atypical lymphocytes are not in keeping with this diagnosis. The vascular proliferation and plasmacytoid cells may mimic angio-immunoblastic lymphadenopathy,[17, 18] but the morphologic triad of arborizing vessels, prominent immunoblasts and amorphous acidophilic interstitial material is not observed in Castleman's disease. The hyaline follicles in Castleman's disease may be mistaken for Hassall's corpuscles. However, confusion with thymoma can be avoided by the fact that no keratin is present, the proliferating vessels penetrate the follicles, and the cellular elements are not spindle cells or epithelial cells as seen in thymoma.

Treatment: In cases of Castleman's disease it appears that local excision is curative and no recurrence has been reported. In our Case 1, a major portion of the tumor was removed in a piecemeal fashion be-

cause of the difficult location. In spite of this, it seems that the patient has been cured. One should have no doubts that if complete removal of the tumor is impossible, partial excision to relieve obstructive symptoms may be useful. Furthermore, there seems to be no other treatment for the occasionally associated intractable microcytic anemia than surgical excision of the tumor.

Acknowledgement

Dan Bergen assisted with photography.

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Rare Infantile Osteomyelitis

Report of a Case Involving the Ribs

RICK WINDLE, M.D.* *and* MEDO MIRZA, M.D.,† *Wichita*

A rare case of infantile osteomyelitis is reported and discussed. The literature is reviewed.

OSTEOMYELITIS of the rib in an infant or child appears to be quite infrequent. A search of the literature reveals only one recent report where tuberculosis was not the causative agent. In that case, the osteomyelitis was secondary to chronic granulomatous disease of childhood. Presented here is a case of a previously healthy 9-month-old male who developed chronic osteomyelitis of the rib. The apparent pathogen was *Staph. epidermis,* coagulase negative.

Case Report

A 9-month-old male presented at Wesley Medical Center on July 23, 1976. Seven weeks prior to this admission he was seen by another physician who surgically excised a 1-cm lump on the right side of the anterior chest wall. The mass was tender, indurated and inflamed, and the pathological report was compatible with abscess.

Following the operation, the wound had not healed and was discharging a seropurulent fluid. Significant negatives included no history of recent infection, trauma, bruising, or systemic symptoms. There was no history of journeys, pets, or exposure to foreign travelers.

Past medical history revealed a full term baby weighing 3 kg 150 gm at the time of birth. Pregnancy was without complications. The patient was current on immunizations and had experienced no previous hospitalizations or significant health problems. The family history provided no useful information.

Physical findings at the time of admission included a draining sinus of the right side of the anterior chest wall overlying the fifth rib, with some mild inflammation and moderate induration. No lymphadenopathy was noted. The rest of the examination was normal.

Laboratory findings were as follows: hemoglobin, 11.6 gm/100 ml; MCV, 70 per cent (normal for age, 77%); urinalysis, 3-5 WBC; WBC, ESR, quantitative immunoglobulins, and total protein, normal; TB skin test, negative.

X-ray examination on July 23 and 25 showed osseous abnormality of the fourth, fifth and sixth ribs, with the most severe changes in the fifth rib. These changes were noted as thickening of the cortex and expansion of the medullary cavity sufficient to double the diameter of the fifth rib. Roentgenograms obtained on June 14 outside the hospital were similar in appearance. The radiologist diagnosed osteomyelitis, chronic in nature. Skeletal survey and IVP were normal.

Clinical Course

The patient exhibited a mild fever of 37.7C during the first 96 hrs following admission. No specific therapy was initiated. The patient's failure to show improvement together with the x-ray findings resulted in the scheduling of surgery on the fifth day following admission.

At operation, a resection of the fifth rib was performed. The rib exhibited numerous abscess cavities toward its anterior aspect, and these were extremely adherent to surrounding periosteum. The operative bed was drained.

The pathological report listed evidence of chronic osteomyelitis. All special stains were negative. Fungal cultures × 2 were negative. Acid fast × 2 showed no growth. Aerobically, *Staph. epidermis,* coagulase negative was grown.

Postoperatively, the patient was treated with aqueous penicillin until the return of sensitivities. On the second day, the regimen was changed to Staphcillin, 125 mg/6 hrs. The child's postoperative rectal temperature of 39.4C decreased to 37.7C by the third day, and was normal by the fifth day.

Ten days following surgery, the patient was afebrile and in improved condition. The drains and sutures were removed and he was discharged. The

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Staphcillin regimen was continued until the 14th day. There has been no recurrence of the disease.

Comment

The infant's presentation was interesting, but not unusual. No specific triggering event could be determined. Weissburg,[1] in her paper on osteomyelitis in the neonate, accounted recent infection more often than trauma to be the preceding event. Nade[2] extended this concept to the infant and child, although trauma in the older ehild was most often predisposing. Both agreed that many times no causative factor could be isolated.

More unusual was the location of the infection in the area of the ribs. In the retrospective report from the Mayo Clinic, Morrey and Peterson[3] reported the femur and the tibia as the most common locations in more than 70 per cent of 135 cases. This compared with similar papers and reviews.[2-7] Bujak and Chusid[8] reported a case of osteomyelitis of the rib in an immune-depressed ehild with chronic granulomatous disease. None of these papers specifieally mentioned infectious involvement of the ribs in a healthy infant.

In assessing the offending pathogen, Morrey and Peterson[3] reported six cases in their series where the agent was *Staph. epidermis*, coagulase negative. In light of three positive cultures taken from the wound pre-, intra-, and postoperatively — all growing coagulase negative *Staph. epidermis* — it appears this was the likely causative agent. This was also supported by the resolution of the fever after the institution of Staphcillin, to which the *Staph. epidermis* was shown to be sensitive.

Due to the lengthy symptoms, resolution without recurrence was propitious. Several of the articles reviewed indicated this was less likely with the chronicity of disease presented here. Surgical excision and drainage coupled with postoperative antibiotics appropriately sensitive were the ingredients of successful treatment.

Conclusion

The presentation of chronic osteomyelitis of the fifth rib in an infant is rare. Also, isolation of *Staph. epidermis* as the likely offending agent is uncommon. Resolution of the problem without recurrence after therapy is fortunate.

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Hyperthyroidism and Periodic Paralysis

Hypokalemia in a Black Patient

GAROLD MINNS, M.D.,* NANCY S. NOWLIN, M.D.,† *Wichita*
and HOWARD DAY, M.D.,‡ *Kansas City, Kansas*

HYPOKALEMIC periodic paralysis is a dramatic clinical entity that may be sporadic, familial, associated with hyperthyroidism, or a result of potassium loss. The hyperthyroidism-associated form is most frequently seen in Oriental patients. There is one previously reported case in a black patient.[1] The following is a case report of a 24-year-old black male who presented with hypokalemic periodic paralysis, followed three months later by hyperthyroidism.

Case Report

In June 1977, a 24-year-old black male was brought to the emergency room complaining of paralysis. The previous day he had eaten 10-15 pieces of fried chicken, a hamburger, and two pieces of apple pie at one sitting. That evening he had noted tingling and weakness in his legs. He had awakened the next morning unable to move and was found by friends. He had noticed mild transient episodes of weakness in his legs two or three times in the previous month; he denied weight loss, tremors, drug use, licorice ingestion, knowledge of tick exposure, renal disease, vomiting, diarrhea, or family history of similar episodes of weakness. His general health had been excellent and he had been on a college football team.

Physical examination revealed the following: the patient was afebrile, with blood pressure, 160/90 mm Hg; pulse rate, 92 and regular; respirations, 20. The head, chest, heart, and abdomen were normal. There was no thyromegaly. Neurologic examination revealed nearly complete symmetric flaccid paralysis of extremities. Deep tendon reflexes were diminished. Sensation, cranial nerves, ocular fundi, and plantar reflexes were within normal limits.

The serum potassium level was 1.4 mEq/L, and the patient was treated with intravenous fluids containing potassium chloride. Twelve hours later, the serum potassium level rose to 2.6 mEq/L, and the patient's strength was improved. Thirty-six hours after admission, the potassium level was 4.7 mEq/L, and the patient's strength had returned to normal. A search for the cause of the hypokalemia was unrewarding. Laboratory evaluation revealed T_4, 8.2 μG/dl; aldosterone, 0.9 ng/dl; serum cortisols, creatinine and BUN, normal. The patient was thought to have the sporadic form of hypokalemic periodic paralysis, and was dismissed on potassium supplements with instructions to alter his eating habits.

The patient moved to another town and continued to experience episodes of hypokalemia and paralysis, for which he was hospitalized several times. Potassium chloride and spironolactone failed to control the attacks.

This condition, primarily afflicting Orientals, is here reported in a black patient. The case report is presented, the literature is reviewed, and various ramifications of the clinical entity are discussed.

He returned to Wichita in September 1977, complaining of weakness and cramping in his legs following the ingestion of 5.5 liters of cola the evening before. He had lost 23 kg since June. He again denied tremors, drug use, diarrhea, or renal disease. Pulse rate was 92; blood pressure, 110/70 mm Hg. There was no thyromegaly, tremor, proptosis, diaphoresis, or muscle wasting, although he exhibited mild muscular weakness. The serum potassium level was 2.5 mEq/L; it rose to 4.7 mEq/L with acetazolamide and intravenous KCl. A serum T_4, drawn before the patient was discharged, was greater than 24 μG/dl, but the patient had left Wichita and could not be found.

In October 1977, the patient was admitted to the Kansas University Medical Center, where he was found to have an elevated T_4 (18% μG%) and T_3 by RIA (400 mg%). He was treated with I-131. The T_4 returned to normal, the episodes of paralysis resolved, and the patient began to gain weight.

Discussion

Hypokalemic periodic paralysis may be familial,

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sporadic, associated with thyrotoxicosis, or associated with potassium deficiency.

The sporadic, familial, and thyrotoxic forms resemble one another in clinical presentation.[2] Paralysis occurs when the extracellular potassium shifts into the cells, reducing the serum potassium. The attacks may be precipitated by ingesting a meal high in carbohydrate, vigorous exercise followed by rest, anxiety, exposure to cold, or the administration of glucose and insulin. The attacks often begin at night and the patient awakes paralyzed. The paralysis usually involves the muscles of the proximal limbs and some of the trunk; the diaphragm and the muscles supplied by the cranial nerves are rarely involved. Between attacks of paralysis the serum potassium level is normal.

Potassium deficient states, including primary aldosteronism, potassium-losing nephritis, chlorothiazide use and Fanconi's syndrome, have been associated with hypokalemic periodic paralysis. In these forms there is actual potassium loss rather than temporary redistribution.

The familial form has been reported mainly in whites, although two cases of affected black families have been reported.[3, 4] It is thought to be inherited as an autosomal dominant, affecting males more often than females in a ratio of 3:1.[2] Onset is at age 15-30 years. With increasing age, the frequency of episodes of paralysis decreases, but the patient is left with myopathy and permanent weakness of mild to moderate severity.

Hypokalemic periodic paralysis is also associated with hyperthyroidism. This has been reported most frequently in Oriental patients; one previous case in a black patient has been reported. In one study of 6,333 Oriental patients with hyperthyroidism, the incidence of periodic paralysis was 1.9 per cent.[5] Although there were 5,119 female patients and 1,214 male patients, there were 99 cases (8.2%) of paralysis in male patients and only 20 cases (0.4%) in female patients. The onset of paralysis was most common between the ages of 20-39 years.

Our patient was originally thought to have a sporadic form of hypokalemic periodic paralysis due to his negative family history and normal T_4. However, it is possible for the patient to develop periodic paralysis before the hyperthyroidism is manifest in any other way. In Okinaka's study,[5] 17 per cent of the patients reported that the periodic paralysis began before the hyperthyroidism. However, these data are based on the patient's recollection, and laboratory studies are not available. Layzer and Goldfield[6] reported a case of a man of oriental ancestry who developed periodic paralysis while ingesting thyroid extract, the equivalent of 2 gr/day. He had no symptoms of thyrotoxicosis, and T_4 and T_3 were normal. When taken off the medication the paralysis resolved, and when rechallenged it recurred.

Another possibility is that our patient may have had T_3 toxicosis, since T_3 was not measured initially. However, within four months he had an elevation of both T_4 and T_3, and the T_3:T_4 ratio was not particularly high as it usually is in T_3 toxicosis. Ivy *et al.*[7] reported a case of a 19-year-old male (race unspecified) who had periodic paralysis associated with an elevated T_3 and normal T_4. This patient had no symptoms of hyperthyroidism except for consuming large amounts of food without weight gain, and having 6-12 bowel movements daily. The paralysis resolved completely after treatment with I-131.

Treatment of hyperthyroidism-associated paralysis consists of treatment of hyperthyroidism. The familial form has been treated with potassium supplements, spironolactone, and low carbohydrate diets. More recently, acetazolamide has been used with some success. Although the mechanism of action is unknown, one postulate is that by inducing a mild metabolic acidosis with reduction of intracellular potassium, a rapid shift of potassium into the cell is less likely to occur.[8]

Summary

A rare case of hyperthyroidism-associated hypokalemic periodic paralysis occurring in a black male has been reported. It illustrates the point that the paralysis may precede any other clinical or laboratory evidence of hyperthyroidism.

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PMMA

Treatment of Pathological Fractures Using Methylmethacrylate To Enhance Strength of Internal Fixation

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Methylmethacrylate was used as an adjunct to metallic internal fixation in treating pathological fractures. The technique effectively facilitated stability in extensively damaged involved areas. All patients obtained relief from pain and some return of function, and there were no adverse complications due to the use of cement. Survival time was not predictable.

A PATHOLOGICAL FRACTURE produces severe complications for patients with malignant disease. Combined with the debilitating presence of cancer, such fractures often cause greater than normal pain and incapacity, and may condemn the patient to bedrest and hospitalization for the remainder of his days. Unfortunately, these fractures are quite common. Autopsies have shown that skeletal metastases probably occur in 70 per cent of patients dying from malignant tumors.[1] Recent improvements in chemotherapy and radiotherapy will prolong life expectancy, but lead one to expect increasing numbers of patients suffering fractures through metastatic lesions.

These fractures present difficult problems in terms of adequate fixation, primarily because of the extensive bone destruction which is always greater than appears on x-rays. Standard methods of internal fracture fixation can be applied to a pathological fracture, but bone destruction is often such that the stability achieved is inadequate.

Recently, Harrington *et al.*[2] demonstrated the effectiveness of polymethylmethacrylate (PMMA) bone cement in augmenting the standard methods of internal fixation for pathological fractures. The physical properties of acrylic cements have been studied since 1938, but it is only within the past five years that the FDA has permitted its general use. In its rubbery visco-elastic state, PMMA can be forced into the medullary canal and molded into the cancerous defect of the bone where it self-cures into a hard mass, strengthening the involved bony area and enhancing the stability of the prosthesis. Thus, the cement substantially improves the rigidity of fixation and distributes the stress on the weakened bone over a greater area.

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The purpose of this disucssion is to evaluate the use of PMMA as an adjunct to internal fixation in the treatment of pathological fractures. The pathological fractures reported here were treated in four Wichita hospitals between 1973 and 1977.

Materials and Methods

From February 1973 through March 1977, 15 patients with 16 pathological fractures underwent internal fixation employing methylmethacrylate cement to increase the stability and rigidity of fixation. The group consisted of ten females and five males ranging in age from 48 to 80 years. The femur was involved in 12, the humerus in 4 and 1 was bilateral. *Table I* shows primary tumor incidence.

TABLE I

PRIMARY TUMORS

Breast	
Female	4
Male	1
Multiple myeloma	4
Lung	3
Renal	2
Endometrial	1
Squamous cell	1

TABLE II
METHODS OF FIXATION

Humerus	Rush Rods	4
Femur	Plates	3
	Zickle nail	1
	Wesley Street	1
	Kuntscher	3
	Jewett	2
	Gilberty	1
	Charnley-Mueller	1

The fractures were treated to alleviate pain as soon as the patient's medical condition allowed. The patient was returned to his home environment and family for his last few months. The specific method of fixation depended upon the type and location of fracture and the surgeon's preference *(Table II)*. There was extensive involvement of the femoral head and neck in one case, and a long stem (Gilberty) prosthesis was necessary. A total hip replacement was used in one patient with extensive femoral head involvement; the acetabulum was not involved.

Early Complications

Two patients had serious postoperative complications. One developed postoperative hypotension and decreased renal output which was followed by a urinary tract infection and an episode of atrial fibrillation. He also developed a large sacral decubitus and a weeping ulcer of the anterior chest caused by his breast carcinoma. The other patient became paraplegic secondary to cerebral metastases one-two months after surgery.

Less serious problems developed in other patients. One patient developed fever of 39.4C, which resolved rapidly with pulmonary toilet and antibiotics. Low grade fever occurred in three patients but resolved spontaneously. Three patients developed sterile serosanguinous drainage which ceased spontaneously in a few days. One patient with a proximal humeral fracture developed massive postoperative swelling of the arm which subsided rapidly upon elevation of the arm. This patient also had sterile serosanguinous drainage which was present postoperatively and was still present at the time of dismissal two weeks later. This was probably due to tissue necrosis in the large metastatic tumor which was being treated with cobalt therapy. An infection developed after the patient returned home. She reentered the hospital two weeks later for incision and drainage of the arm and removal of bone sequestra



Figure 1. M.G., 68, cancer of the breast.

Figure 1-A. Lytic lesion of the femoral neck.

and a few fragments of cement. The fracture remained stable and the patient was able to continue her home treatment.

Failures

There was one failure of fixation due to unknown causes. This occurred in a 64-year-old female with metastases to the femoral shaft. The IM rod "backed out" after nine months and required reinsertion and fixation employing screws and wire. The rod again loosened two years later and is causing the patient some pain. X-rays show loosening around the distal end of the rod. The patient has declined further surgery and when last seen was ambulating with external support within the home.

Results

All patients had marked pain relief as a result of their fracture fixation, although some had pain due to other metastases. Patients with humeral fractures achieved a useful range of motion and function of the affected limb. All patients with femoral fractures were substantially relieved of pain and were sitting in a chair within a few days after surgery. Four were walking at the time of discharge with partial or full weight-bearing; two others who were severely weakened from disseminated disease were also able to leave the hospital.

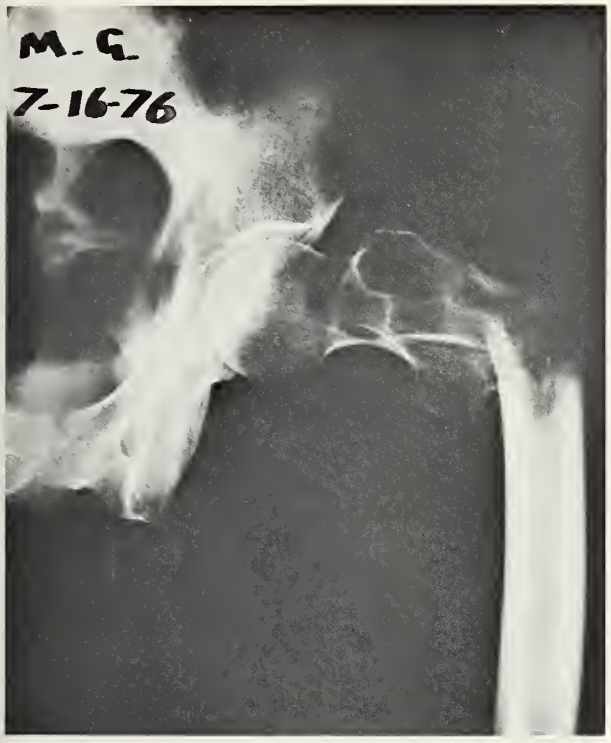


Figure 1-B. Pathological fracture one month later.

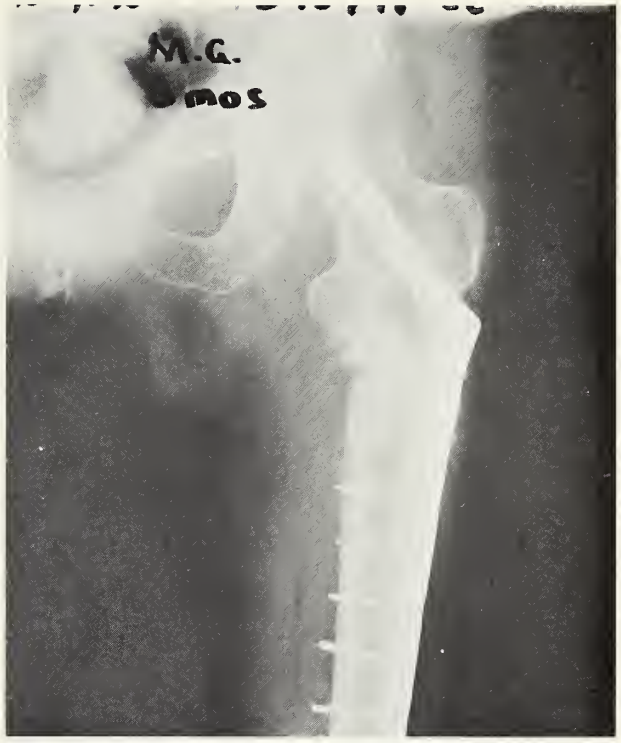


Figure 1-C. Jewett nail with cement.

Survival

The survival time following fracture fixation ranged from three weeks to four years, averaging eight-nine months. One patient survived only three weeks as the disease proved far more extensive than was thought. Three patients survived one-three mos, two survived four-six mos, and four survived seven-twelve mos. To date, survival times of two years and four years have been achieved by two patients with primary kidney involvement.

Discussion

Apart from the use of the total hip prostheses and the incorporation of methylmethacrylate, the methods of fixation were no different from those used to treat non-pathological fractures. The two total hip prostheses were used because of extensive bone destruction of the head and neck of the femur or of the interochanteric region. In these cases, the removal of the tumor tissue would have left insufficient bony stock to allow adequate fixation with conventional hip nail-plates. Extensive acetabular involvement is a contraindication to using a total hip prosthesis, because of difficulty of achieving stability for the cup and the possibility of migration of the cup into the pelvis.

The methylmethacrylate was inserted into these fractures in a semi-fluid stage, rather than in a doughy state as usually used, so that it would mold around the fixation device filling the medullary canal and the irregular defect created by the removal of tumor and necrotic tissue. No hypotension was experienced at the time of cement insertion, although some earlier reports stated this was a problem.

The effect of methylmethacrylate on bone healing has been studied by Charnley,[3] Yablon,[4] and Lindwer.[5] Early reports were not encouraging, but subsequent clinical and experimental work has shown that bony union will occur provided the periosteal surface is left free of cement. Pathological fractures heal slowly, however, and reports differ widely on healing capabilities; Clain[6] reported healing in only 5 per cent of femoral neck fractures, Perez[7] reported 70 per cent of all femoral fractures healed and 100 per cent humeral fractures healed when rigid internal fixation was used, as opposed to 33 per cent and 68 per cent respectively without rigid fixation. Healing in our series appears low, as 25 per cent of the patients died within three months before healing could take place. However, healing occurred in all but one of the long term survivors (six mos or more).

Complications may be expected to occur more frequently when operating on patients with carcinoma and especially those with disseminated dis-

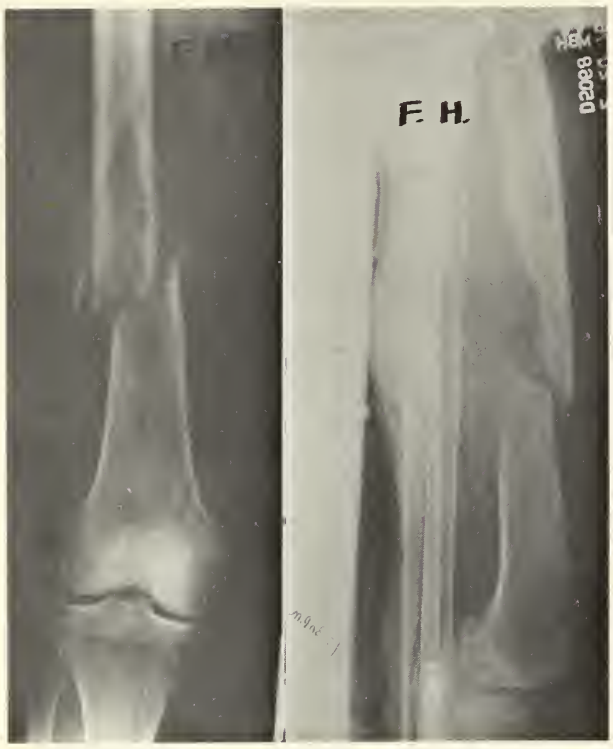


Figure 2. F. H., 78, multiple myeloma.

Figure 2-A. A P and lateral fracture.



Figure 2-B. Postoperative film with plate plus cement.

ease. The majority of the complications we experienced were minor and self-limiting. The deep infection and delayed wound healing in the patient with the fractured humerus may have been caused by the massive postoperative swelling following radiotherapy and chemotherapy which produced tissue necrosis. The postoperative hypotension in the debilitated patient was probably caused by his medical condition rather than the operative procedure.

Failure of fixation when using bone cement[2] is an unusual occurrence. In our series, only one case developed partial failure of fixation when the intermedullary rod backed out of the femur. Despite this, the reduction remained unchanged although it had not yet healed.

Early writers[8] expressed concern over the possible effects of secondary ionizing radiation from metallic devices and PMMA, and a possible protection of the tumor by shadowing due to the mass caused by the fixation device and PMMA. Subsequent studies have shown that methylmethacrylate is neither affected by nor does it interfere with radiotherapy.[9] Most authors recommend surgery for patients with an expected survival of three-six mos, but it is difficult to predict the survival time of patients with metastatic diseases as is illustrated by our patient who died unexpectedly three weeks after surgery. Perez[7] found that 50 per cent of his patients with a pathological fracture survived one year and 25 per cent survived two years. Marcove and Yang[10] reported a poorer survival rate: 34 per cent of patients with one fracture survived over one year and 26 per cent with multiple fractures survived over one year. Only 2 of our 15 patients lived more than one year. Parrish and Murray[11] found the average survival time for patients with all types of tumors was only five-twelve mos. This suggests the presence of multiple fractures should not necessarily exclude surgery which could relieve pain, return some function, and allow a home existence. Each fracture patient must be assessed individually.

In other series, breast carcinoma offered a better prognosis for both healing and survival, but this was not true of our five patients, of whom none lived more than six mos and two lived only three mos following surgery. One would expect survival to be related to the type of primary carcinoma. However, in our series, we were unable to draw any such conclusions *(Table III)*. It is interesting that both patients with renal cell carcinoma survived more than two years. One underwent a nephrectomy prior to the fracture, and the other (whose primary tumor

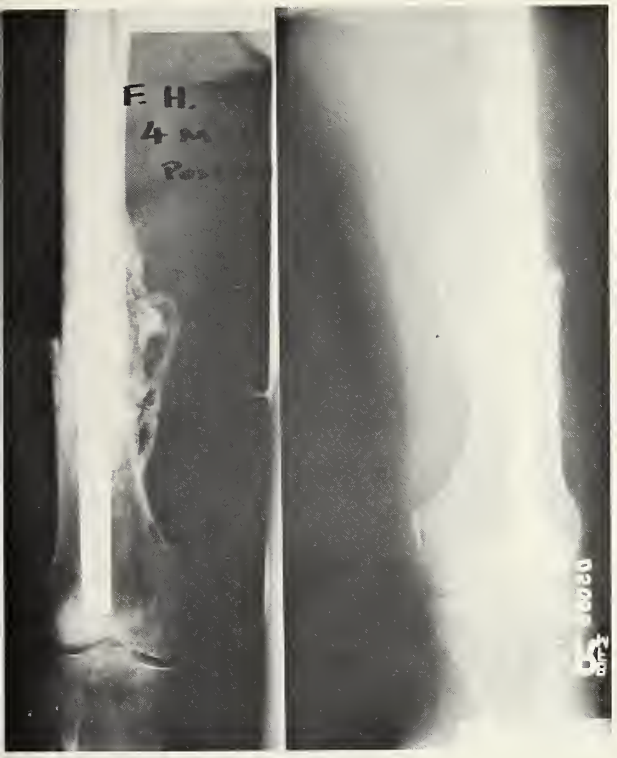


Figure 2-C. Four months postoperative weight-bearing demonstrating early healing.

was undiagnosed until she experienced the femoral fracture) had a nephrectomy following stabilization of the fracture.

The ability to walk is of great importance in restoring the patient's independence. The combined use of PMMA and metal strengthens the fracture site so the bone is more likely to withstand stress. Parrish and Murray[11] reported 21 per cent of their patients were capable of full weight-bearing after surgery using standard fixation methods. Harrington *et al.*,[2] using methylmethacrylate in conjunction with internal fixation, reported that 94 per cent of their group were capable of full weight-bearing. In our series, all but two were ambulating with a walker.

Summary

PMMA in conjunction with internal fixation methods facilitates improved stability, improved bone healing, and a decrease of pain. Ambulation is thus increased, allowing the patient to return home and be less dependent on nursing care and pain medication.

The incidence of pathologic fractures is expected to increase. Correspondingly, the necessity for internal fixation procedure is also projected to increase. It has been shown that PMMA holds metal to bone effectively and safely, and can be used to fill cancerous bony defects. The survival time of metastatic patients is difficult to predict, but with rigid fixation the patient is comfortable and usually able to return home to his family. Therefore, it seems reasonable to consider all patients with pathological fractures and with a life expectancy of three months or more as candidates for rigid internal fixation, unless their medical condition is so poor that they would not be expected to survive the surgery.

TABLE III

SURVIVAL AND TUMOR TYPE

Survival	*Primary Site*	*Patients*
0-1 mo.	Lung	1
1-3 mo.	Lung	1
	Breast	1 F.
		1 M.
4-6 mo.	Breast	1
	Myeloma	2
7-12 mo.	Breast	1
	Myeloma	2
	Squamous cell	1
Over 1 yr.	Kidney	2-2 yrs.
		4 yrs.
Recent alive	Breast	1
	Lung	1
	Endometrial	1

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Current COMMENT

Chronic Hepatitis

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CHRONIC HEPATITIS is one of the unsettled areas in medicine. In recent years there have been rapid advances in methods of diagnosis and treatment of these disorders. Chronic hepatitis is comprised of several poorly defined conditions which differ in cause, course, and outcome. Histologically these disorders all are characterized by prolonged inflammation of the liver that spares the intrahepatic bile ducts. Terminology has been varied and complicated with more than 50 names being used for the same patient populations. In the past, patients with chronic hepatitis were diagnosed on the basis of clinical features, often when the disease was advanced. Many asymptomatic patients have been discovered by unexpected biochemical abnormalities on multiphasic laboratory screening examinations in recent years. It has become obvious that the spectrum of disease is much broader than had previously been recognized. In general, patients with severe morphologic abnormalities are more likely to be symptomatic, but advanced morphologic alterations can be found in asymptomatic patients.

The Fogarty International Convention in 1974 suggested a simplified classification for chronic hepatitis of probable viral or virus-like origin. The suggested designations were chronic persistent hepatitis (CPH) and chronic active hepatitis (CAH). The term chronic aggressive hepatitis is less desirable because this appellation has an unfortunate effect on the patient who surmises that his disease is something akin to cancer. It is important to attempt to distinguish between CPH and CAH, because appropriate therapy differs significantly.

Clinical Findings

CPH is most often characterized clinically by lack of symptoms and apparent good health. Biochemically, these patients have persistent or recurrent, but generally mild increases in serum transaminase levels with complete resolution commonly occurring in six months to two years. CPH is usually less likely to progress to cirrhosis than CAH. Patients with CPH should not receive immunosuppressive therapy, but rather should be followed carefully for at least a year to assure proper diagnosis and lack of progression to a more severe lesion. If biochemical abnormalities have persisted or the patient is asymptomatic, a repeat liver biopsy should be done at that time.

CAH is characterized by more symptomatic patients. Complaints may be vague and nonspecific, such as malaise, anorexia, and amenorrhea. Extrahepatic manifestations are much more common than in CPH, and include arthralgias, Sjogren's syndrome, skin manifestations such as erythema nodosum and spider nevi, as well as other systemic abnormalities. Serum transaminase and gamma globulin levels tend to be higher in CAH than in CPH. Smooth muscle antibodies are found in 66 per cent, ANA in 50 per cent, and antimitochondral antibodies in 25 per cent. CAH is more likely to progress to cirrhosis than CPH, but in some cases

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this progression may be limited by immunosuppressive therapy.

Causes

There are multiple suspected causative agents in CAH. Viral agents are prominent with Hepatitis B having an established association. Hepatitis A is probably a rare cause; however, other viruses which are poorly characterized at the present time are believed to play an important role. These viruses are referred to as non A, non B viruses. There is a growing list of drugs which are suspected offenders; alphamethyldopa and oxyphenasatin are commonly associated with CAH. Wilson's disease should be excluded in patients with CAH, especially those under age 30, because this condition is potentially ameliorated by penicillamine therapy.

Diagnosis

Differentiation between CPH and CAH can be difficult. Stumbling blocks in diagnosis include disagreement in interpretation of the biopsy specimens and sampling error. Histologic findings of cirrhosis may be missed in 40-60 per cent of patients on blind percutaneous liver biopsy. There may be some tendency for transformation from CPH to CAH. Diagnostic laparoscopy may be helpful in yielding more precise diagnoses in patients with clinical laboratory findings suggestive of chronic liver disease, but in which an initial percutaneous liver biopsy shows normal or minimally altered liver tissue. In addition to gross morphologic abnormalities that can be observed laparoscopically, directed liver biopsies can be obtained.

Microscopically, CPH is characterized by lymphoid and plasma cell infiltration of the portal tracts with maintenance of lobular architecture and preservation of the limiting plate of hepatocytes. Minimal lobular scarring and parenchymal inflammation is usually present, although scattered discrete intralobular lympho-histiocytic foci may sometimes be found. A characteristic "cobblestone" arrangement of the hepatocytes which results from regenerative activity is usually present.

CAH is characterized by more destructive lesions. The limiting plate is violated by inflammatory cells that extend into the parenchyma and engulf groups of hepatocytes in a process called piecemeal necrosis. There may be distortion of lobular architecture, bridging necrosis extending from portal tract to portal tract or to central vein, fibrous spur formation, and cirrhosis. Hepatitis B virus sometimes gives hepatocytes a characteristic "ground glass" appearance. The presence of Hepatitis B can be further demonstrated with special stains and immunofluorescence.

Proper timing of liver biopsy is important. Patients with recurrent or persistent elevation of serum transaminases after acute hepatitis should be observed for six months before a liver biopsy is obtained. Piecemeal necrosis can be found in the resolution phase of acute hepatitis in patients who do not subsequently develop CAH.

Hepatitis B and CAH

Patients with CAH are often divided into two groups — those who possess the Hepatitis B surface antigen (HB_sAg) and those who do not. Approximately one third of CAH cases are associated with previous Hepatitis B infection. The preceding hepatitis may have been mild or even anicteric and unrecognized. Sherlock feels that patients with such an inauspicious initial illness may be even more likely to develop CAH than patients with more impressive acute hepatitis. An active inflammatory reaction and subsequent resolution may indicate a more capable immunologic system. People who are maximally exposed to Hepatitis B — such as hospital workers, drug addicts and homosexuals — are at a greater risk than the general population of developing Hepatitis B related CAH. Redeker has published data from the study of 429 patients recovering from acute Hepatitis B infection. Ninety per cent recovered completely while 7 per cent developed CPH and 3 per cent CAH. HB_sAg positive CAH populations tend to have more males, an older age group, fewer associated autoimmune phenomena, and a less certain response to immunosuppressive therapy.

Due to recent improvement in immunologic techniques, various viral antigens and antibodies can be detected that can aid in evaluation of HB_sAg positive CAH patients. Indicators of disease chronicity include persistence of HB_sAg for longer than 12 weeks, and detection of DNA polymerase, "e" antigen and Hepatitis B core antigen (HB_cAg). Magnius and Nielson have shown that the presence of "e" antigen identified patients destined to have more active and progressive disease courses. In addition, "e" antigen seems to be a marker for viral infectivity, and is thus particularly important in evaluating individuals such as health care workers who could potentially infect patients they come in contact with. HB_cAg has recently also been shown to be helpful in predicting prognosis in these patients.

HB_sAg positive CAH patients have been said to have a more benign course than HB_sAg negative patients. This may be due in part to early series tending to include more advanced forms of HB_sAg

negative CAH than was found in the HB_sAg positive CAH group. Schalm has shown that HB_sAg positive patients treated with corticosteroids do worse than HB_sAg negative patients. Immunosuppressive therapy in patients with HB_sAg positive CAH may further impair viral clearance. Clinical trials are in progress to further assess the role of immunosuppressive therapy in the HB_sAg positive CAH population.

Therapy

Corticosteroids. Immunosuppressive therapy has been employed in patients with CAH in the belief that the disease is autoimmune in origin. There have been four major series of patients with CAH treated with immunosuppressive therapy reported in the literature. These studies indicated that corticosteroids were beneficial in the treatment of CAH. Subsequently these studies have been criticized for defects in experimental design, and in addition, certain subgroups, notably HB_sAg positive CAH and patients with mild CAH, were not adequately studied. It has become evident that there is considerable risk of serious side effects associated with prolonged corticosteroid therapy. Such side effects were found in 60-70 per cent of patients in a Mayo Clinic study. Corticosteroid therapy seems to be strikingly beneficial only in more advanced lesions such as bridging necrosis, submassive necrosis and cirrhosis, while responses in mild CAH are less clearcut. In a Mayo Clinic study, six of seven untreated patients with mild CAH did not show progression to more severe lesions. With the recognized danger of serious side effects associated with corticosteroid therapy, it is felt by some that patients with mild CAH should be followed carefully without initial immunosuppressive therapy. Clinical trials to better evaluate treatment in these patients are in progress. It is advisable to avoid immunosuppressive therapy of HB_sAg positive patients with mild or moderate CAH until more definitive information is available from clinical trials. Immunosuppressive therapy of severe HB_sAg positive CAH is usually initiated with 40-60 mg of prednisone or prednisolone daily. After a period of approximately one month the dosage can be tapered to 10-20 mb/day. Alternate day therapy is not as effective as daily administration in the treatment of CAH. At least a two-year course of therapy is recommended. If there is biochemical remission at that time, corticosteroids can be discontinued with surveillance for evidence of recrudescence of the disease. Relapses are treated with reinstitution of the treatment regimen. Remissions in CAH are characterized by absence of symptoms, return of serum transaminases and globulin levels to normal, and a liver biopsy without active inflammation.

Azathioprine. Azathioprine as an adjunct to therapy may be helpful in some patients, but used alone it has been shown to be no better than placebo. In combination therapy, however, it sometimes allows the corticosteroid dosages to be reduced. Azathioprine can be considered for use in patients with excessive corticosteroid side effects, and in patients who have not responded to corticosteroids alone. With liver dysfunction there is altered metabolism of Azathioprine which can sometimes lead to toxic levels of metabolites. Azathioprine toxicity may mimic the natural progressive course of CAH and thus must be used with caution.

Experimental Therapies. Recently various experimental therapies have been attempted primarily in patients with HB_sAg positive CAH. Interferon has been demonstrated to aid in clearance of HB_sAg and to decrease levels of DNA polymerase and HB_eAg in patients with HB_sAg positive CAH. Transfer factor has not proven to be of therapeutic benefit. Levamisole, a drug that seems to act by stimulating cell mediated immunity, holds some promise in future treatment modalities.

Prognosis

Prognosis is good in most patients with CPH, with many having complete resolution. In CAH the course is more variable. Most patients have long periods of biochemical quiescence, with some eventually dying of portal hypertension or hepatocellular carcinoma. Others have a course characterized by periods of quiescence interrupted by episodes of acute liver cell necrosis. A small number (3%) have rapid progression to cirrhosis and death from liver failure within one year. This latter group must not be confused with severe acute viral hepatitis with submassive necrosis that frequently has a rapid downhill course in elderly patients.

In conclusion, modern advances in methods of diagnosis and treatment of chronic hepatitis have led to a more optimal approach to these patients. It is likely that further improvements will be forthcoming.

Self-Assessment Questions

True or False

1. Patients with chronic persistent hepatitis should receive immunosuppressive therapy to prevent progression to cirrhosis.
2. Hepatitis A is not felt to be an important causative agent in chronic active hepatitis.
3. Patients should have a liver biopsy soon after an

acute hepatitis episode to predict progression to chronic hepatitis.

4. Patients who have HB_sAg+CAH should receive the same therapy as HB_sAg negative patients.
5. Daily rather than alternate day corticosteroid therapy should be utilized in patients with CAH.

(Answers on page 521)

Acknowledgements

Dr. Allan R. Cooke helped review the manuscript. Stephani Johnson provided technical assistance.

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Breast Surgery

(Continued from page 487)

on top of the Silastic implant, and a breast mass can be detected by the patient and examined by her physician. Augmentation surgery of the breast does not interfere with mammography, and may improve the procedure in some patients as the breast is projected away from the chest wall giving a better image on the x-ray film.

During pregnancy, the normal breast overlying the implant can enlarge without difficulty, and lactation is not affected by the surgery. Subsequent to augmentation, many patients have undergone enlargement of the breast without difficulty during pregnancy.

At this time, the long term results appear to be good without undue ptosis or other breast change.[8] The procedure has been greatly simplified by local anesthesia and outpatient surgery, and the convalescent period in most cases has been shortened to a few days.

Summary

The results of the most recent 300 cases of augmentation mammoplasty have been reviewed to identify the types of patients and types of problems encountered in those who seek breast enlargement. The management of these patients as well as the postoperative complications were examined and discussed.

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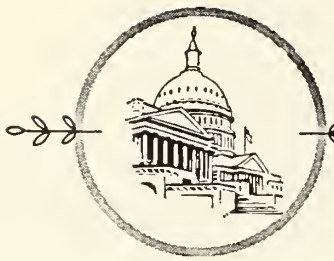
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Socio- ECONOMICS

Retirement Plans: Self-Employed vs Corporate

THE MOST SIGNIFICANT of the tax advantages to be gained by incorporating is the difference in treatment of corporate pension plans versus the self-employed retirement plan. The self-employed plans (sometimes called Keogh plans) are subject to numerous restrictions and do not receive benefits available to professional corporations. These restrictions were formerly more extensive than they are now, but are still significant. The main differences are illustrated below.

How Much Can Be Set Aside

A professional covered by a self-employed retirement plan may deduct for income tax purposes 15 per cent of earnings, with a limit of $7,500 per year. He must also contribute the same percentage for his employees. However, a corporation can put aside for an employee 15 per cent of compensation paid to a profit-sharing plan. If both a profit-sharing and a money purchase pension plan are adopted, up to 25 per cent can be set aside on a favorable tax basis. There is an overall limit on company contributions to retirement plans, which increases each year, adjusted for inflation. In 1977, the limit was approximately $28,000 for any one individual's account. Most who are aiming this high will utilize a combination of profit-sharing and pension plans because of the flexibility provided by the profit-sharing plan.

Taxpayers in high brackets may want to set aside more than $7,500/year, and there are many, especially professionals, who may want to exceed that limit. The higher the individual's income and the larger the percentage set aside, the more spectacular are the differences in the amount which a person may accumulate for his retirement. For example, $7,500/year set aside at 6 per cent for 15 years produces about $185,000, while $28,000/year set aside at 6 per cent for 15 years produces about $675,000. A difference of nearly $490,000 is thus accumulated in the corporate plan.

Integration with Social Security

In a self-employed retirement plan, if more than one-third of the benefits accrue to the self-employed, there can be no consideration of employer social security tax payments as a feature of the plan. In such cases, the same percentage contributed to the retirement plan for the professional must also be contributed for the employees.

Example:

	Income		*Contribution*
Physician	$50,000 × 15%	=	$7,500
Nurse A	9,000 × 15%	=	1,350
Nurse B	8,000 × 15%	=	1,200

In a corporate plan, however, no such requirement exists. A corporate retirement plan may consider as part of the cost (and benefits) the social security payments, and contribute a larger sum to those portions of salary above a certain limit (no higher than the total being taxed for social security purposes). The maximum differential for a profit-sharing or money purchase pension plan is 7 per cent. Thus a corporation whose shareholder-employees are the only ones on a relatively high salary can adopt a plan providing a contribution of 7 per cent of the excess above a certain level of salary — say $10,000 — and nothing on the first $10,000. Such a plan would benefit only the highly paid, but is not considered to be discriminatory because of the employer social security tax contributions. Any payments in excess of the permitted differential are usually assigned pro rata, in proportion to compensation, to all plan participants.

Example:

	Income		*Contribution*
Physician	$50,000 × 25%	=	$12,500
Nurse A	9,000 × 18%	=	1,620
Nurse B	8,000 × 18%	=	1,440

Vesting

An even more significant savings may accrue to

the professional who incorporates as a result of vesting or ownership rights in the pension or profit sharing plan.

While the Internal Revenue Code requires that all monies set aside in a Keogh account for an employee become their property immediately, no such restrictions apply to the incorporated professional.

Depending on plan design and other factors, 10 years of service may be required before any ownership rights accrue to the employee. In the event of the employee's termination prior to completion of this 10 years of service, all monies the professional corporation has contributed for him are returned to the corporation.

Defined Benefit Pension Plans

Still larger amounts of up to $75,000, depending on age and income, can be set aside in certain situations in a defined benefit pension plan because such a plan is limited as to benefit payments but not to the amount contributed. A special rule also allows greater contributions for a money purchase pension plan used in combination with a defined benefit pension plan.

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Chronic Hepatitis

(Continued from page 519)

Answers

1. *False.* CPH is generally a nonprogressive disorder that is unlikely to progress to cirrhosis. These patients should be followed carefully until at least biochemical resolution has been present for one year.
2. *True.* Hepatitis B does have an established association, but Hepatitis A does not appear to be an important causative agent in CAH. Other poorly characterized viruses referred to as Non A, Non B viruses may be involved in some cases.
3. *False.* In most cases it is preferable to wait for six months following an acute hepatitis episode before doing a liver biopsy in patients suspected of chronic hepatitis. Biopsies obtained sooner are sometimes difficult and confusing to interpret.
4. *False.* Immunosuppressive therapy should probably be avoided in patients with HB_sAg positive CAH because it may further impair viral clearance. Clinical trials are now in progress to more completely assess efficacy of immunosuppressive therapy in these patients.
5. *True.* Alternate day corticosteroid therapy has not been found to be as effective as daily administration.

TIME MANAGEMENT & APPOINTMENT SCHEDULING WORKSHOPS

A smoothly run medical office is one where patients are seen at the appointed time. Unfortunately, high on the list of patients' complaints regarding their contacts with the physicians is the long wait in the office.

To assist physicians in streamlining their appointment scheduling procedures, the Kansas Medical Society, in cooperation with the American Medical Association, will present a series of workshops geared to the PHYSICIAN and the APPOINTMENT SCHEDULER.

To enable your Medical Assistant to derive the full benefit of this educational effort — and because the physician is an integral part of the appointment — it is important that You, Doctor, attend this half-day workshop with your Medical Assistant.

Registration fee is $25/person. Attendance is limited to 50 persons at each session. Please respond to the announcement promptly when it reaches your office. Scheduled sites are as follows:

TOPEKA Holiday Inn — Tuesday, November 14, 1978 — 8:00 AM
SALINA Hilton Inn — Wednesday, November 15 — 8:00 AM
WICHITA Canterbury Inn — Wednesday, November 15 — 6:30 PM
WICHITA Canterbury Inn — Thursday, November 16 — 8:00 AM

How to Start to Stop

Ed Note: This is the seventh in the series of articles prepared by the AMA Department of Practice Management, Division of Medical Practice. For other articles on this topic, see the following issues: November and December 1977, and January, February, March, and July 1978.

The subject of practice management has become more critical in recent years. To assist the physician in a smoother operation of his office, the Department has produced workshops for medical assistants dealing with such aspects as collections, public relations-telephone techniques and office management, as well as practice management workshops for the new physicians. The Kansas Medical Society Executive Office will present such workshops upon demand by members.

THERE IS A VICARIOUS thrill in reading the occasional news story about "Old Doc" who started practice in a one-room walk-up 60 years ago and today, at 92, is still going to the office every day to serve the health needs of the community. However, fewer physicians today are practicing until they "die in the harness." There are a number of reasons for this in addition to the major fact that the physician, as is true of any other individual, is entitled to retire and enjoy the fruits of long years of arduous service to society.

Today (and this wasn't always the case) the physician is covered under the Federal Social Security program and, in addition, is allowed to establish a tax deferred Keogh, Corporate, or IRA Retirement Plan. He no longer has to depend on after-tax savings to finance living expenses and the things he would like to do after he ceases to practice.

A recent and more dramatic reason for an increasing number of physicians retiring from active practice is the professional liability insurance situation. Malpractice insurance costs have doubled and tripled with no relief from the nearly confiscatory costs for the older physician who might want to reduce his workload to an acceptable level for his aging years. Insurance protection is needed whether he practices two days/week or 65 hrs/week.

A third factor is the sociological demand to keep up-to-date in an era of rapid change. Patients no longer automatically consider "Old Doc" the very best because he has been in practice for 30 years. They sometimes question whether he has the knowledge and expertise of younger physicians more recently graduated from medical school. And his colleagues on the hospital staff are requiring stricter adherence to eligibility standards for medical and surgical procedures.

There is a considerable amount of work involved in closing a medical office. Before you can walk away from your practice, you must consider:

Informing your patients;
Advance notice to your employees;
Transferring medical records;
Medical record retention;
Medical insurance claim forms that come in after you have locked the door;
Cancellation of business insurance policies;
Your narcotics license;
Disposition of narcotics and medical supplies;
Rent, lease contract, disposition of real estate;
Office equipment and furnishings;
The telephone;
Membership in various associations;
Magazine subscriptions;
Business bank account;
Taxes;
Keogh or pension program involving employees.

In addition, you will want to carefully review your own insurance program, stock portfolio, other savings and investments, and any real estate situations in which you may be involved.

You may also want to arrange for continued medical care for yourself and your family, perhaps including a medical-hospital insurance program.

Advance planning should start a minimum of *two years ahead* of the closing date. Many of the things you do will have to be accomplished in the last three months and even later, but you can be prepared to do them. For example, you may wish to give your patients a three-month notice, assuming a general type practice. The letter you will send to active patients can be prepared in advance. Your files can be checked for the "actives" and "inactives." For patients who indicate to you that they want their medical record information transferred to another physician, you can plan the best way to do this. (You will want to keep your records, of course, and forward only summaries or Xerox copies.)

When and how to give notice to your employees is an individual situation problem. You will need adequate help in your office to the last days of practice and probably some assistance for some time thereafter. If you have career employees who want or need to continue to work, they will logically start looking for new positions as soon as they find out that you have definitely specified a closing date. Perhaps you can arrange for a key person to stay with you until the end, or find out if temporary help is available in your area to fill in any necessary spot that becomes vacant.

You may find it advantageous to set a closing date to coincide with your rent or lease contract. You should check ahead with your county medical society or medical equipment distributor to find sources

available for disposing of your office equipment, medical books, etc. Once the door is locked and the lease cancelled, these things have to be moved, and putting them in storage is at best a temporary solution.

Advance notices to the societies in which you are a member are in order, and the cancellation of magazines which you no longer want to receive, or address changes for those you want sent to your home, should be made several months in advance. If you are approaching the age when you will receive Social Security, you will want to contact the SSA office several months in advance of your 65th birthday.

Business insurance can be cancelled from the date you close the office, and refunds may then be requested from the unexpired insurance. If you have a retirement or Keogh plan that includes employees, they must be advised of the amount of their vesting and how these funds can be transferred. Your malpractice insurance should be maintained until the very last day of your practice, and you should locate and maintain a copy of every malpractice insurance policy issued to you during the years of your practice.

Make every effort possible in the last three months of practice to collect outstanding receivables. The collectability of old accounts declines radically after the office is closed. You might well consider turning uncollected accounts over to a medical collection service immediately after the closing date. The collection service should receive them quickly in order to achieve recovery for you.

Physicians who by choice have practiced alone for years may wish to acquire a partner for the last year or two of their practice, with the purpose of providing uninterrupted medical service to patients, as well as to have a ready buyer for their practice when the retirement date finally arrives. And, while patients appreciate the opportunity of gradually adjusting to a new physician, the new physician will also benefit from being introduced into an established practice, since this method typically results in high patient retention.



THE MENNINGER FOUNDATION

Continuing Medical Education

Understanding Hypnosis: Sept. 11-12, 1978. Directed by Dr. William Trussell.

Effective Techniques for Medical Teaching—featuring Dr. Hilliard Jason: September 23-24, 1978.

Psychopharmacotherapy for Children and Adolescents—featuring Dr. Judy Rapoport: Sept. 25, 1978.

Rehabilitation: A national conference on the psychological aspects of rehabilitation: Oct. 5-7, 1978.

Biofeedback Training: Its Appropriateness: Oct. 14-15, 1978. Directed by Dr. Dale Walters.

CME credit available.

For further information contact June Housholder, Division of Continuing Education, The Menninger Foundation, P.O. Box 829, Topeka, Kansas 66601. 913/234-9566, ext. 3685.

The President's Message

The recent AMA House of Delegates meeting in St. Louis, June 17-23, provided many highlights for Kansans. On Sunday, Dr. Karl Menninger was presented the Rodman E. and Thomas G. Sheen award for scientific accomplishment. The award consists of $10,000 and a commemorative plaque. In his acceptance speech, Dr. Karl thanked American medicine for the honor on behalf of his family and his specialty. He was the only recipient to receive a standing ovation. The lady seated in front of me summed it all up when she said, "What a beautiful human being."

On Monday evening, at the Kansas Dinner for all Kansans attending the meeting, everyone present was thrilled by Dr. Menninger's homespun remarks, captivated by Mrs. Karl's wit and charm. We thank them for staying over just so they could be with us. We were also honored to have Mrs. Irma Schafer and Mrs. Leila May Young in our midst. They are the only Kansans to hold the presidency of the AMA Auxiliary.

Tuesday afternoon, I attended the Auxiliary installation of their incoming President, Mrs. Bergnes, and witnessed the love and affection shown to their retiring President, Leila May. She is truly a wonderful lady, and we can all be proud of her and the job she did in keeping the Auxiliary running smoothly.

During the sessions of the House of Delegates, our representatives, Drs. Clair Conard and Alex Scott, and their alternates, Drs. Lew Purinton and Kermit Wedel, were visible and vocal. Our resolutions fared well; the problems we addressed were given sympathetic consideration and understanding. The resolution sponsored by the Kansas and Oklahoma delegations, to require the AMA staff to present to the House of Delegates any further AMA proposed national health bills prior to the December meeting of the House, was passed. Dr. Bill Reals represented the American College of Pathologists, and Dr. Ralph Hale represented the American College of Allergy.

I know there are some among us who feel animosity toward AMA. "They don't represent me!" "They're too timid!" "They're too aggressive!" "They're selling us down the river to the government!" "They're always opposing the Feds and making us look reactionary." "They had no business submitting an NHI bill." "They didn't go far enough in the NHI bill they submitted."

Sounds like any doctor's room on any given day? As I listen, I am always amazed at the entirely opposite ways that the AMA is viewed by physicians. There are many varied reasons for NOT joining the AMA; some may even be true. It's easy to criticize any organization that you really haven't become involved in, but I think the AMA deserves a close, studied look before you decide against it.

The image of the AMA in the mind of the average American is changing. We are not perceived as the "bad guy," thanks to their current attitude toward government irresponsibility and inactivity, and the overkill attempts by the government and the media to discredit medicine. People are learning NOT to believe everything they read in the paper or see on TV.

The AMA has also become more aggressive. It has taken the federal juggernaut to court and won some of the battles. At least our views are being heard and weighed. Our testimony is being sought more and listened to as the realization of the cost of any NHI program; of MD excess rather than shortage (as predicted by you know who); the "boners" by government (swine flu, saccharine, etc.) have reduced government's omniscience in the public mind.

You should have received a yellow folder through the mail in July, entitled: AMA Status Report Listing Some of the Services to You and Your Patients Provided by AMA. It should receive your attention because, as they say, we can do more together.

The government leaders listen to numbers, and on that argument alone you should consider AMA membership. It is a sad commentary on the political scene, but it is members — not fact, philosophy, or principles — who count.

While we all have our own specialties — and they are indeed valuable — we really decimate ourselves and our influence through this approach. We must be doctors first, and specialists second! The government plan to divide and conquer is evident in the recent suit brought by the FTC against the American Society of Plastic and Reconstructive Surgery. Only through a "big" organization that represents all of the doctors can we hope to defend against such bullyism by big brother. We must unite and stand together. The price of freedom is eternal vigilance, and that means involvement! Government will not stop its attempts to control health care at all levels (and that means us).

It is not necessary to agree with everything an organization does to become a member and stay involved. If that were true, I submit that many of us would not be Americans, married, physicians, or members of a society. Instead, we would be in our own little caves or on a mountain top contemplating our navels!

Fraternally yours,

Warren E. Mayer, M.D.

President



Editorial COMMENT

Back to Bakke

In her engaging account of the distaff side of British royalty, "Queens of England," Norah Lofts introduces the chapter on Queen Victoria with the question, "Is there, indeed, anything left to be said about her?"

The same thought comes to mind in the aftermath of the Bakke decision. The case was scrutinized exhaustively throughout its progress to legal sanctification and the decision has been dissected and analyzed in a manner generally reserved for the marital misadventures of the entertainment world. The patchwork opinion produced by the Supreme Court with its barely decisive majority has produced, in turn, a spate of comments varied in vehemence but predictable by source. Most of the minority spokesmen viewed with alarm, the plaintiff's lawyers pointed with pride, and medical school (and other academic) administrators ventured a cautious sigh of relief. At least, they were told how not to do something even if not too clearly how to do it. And one might think there was nothing left to be said—if one didn't have to deal with the compulsive editorialists. Miss Lofts *did* go ahead and write about Victoria so —

The positive effect of the decision was, of course, the elimination of quotas as a mechanism for increasing minority representation in academic admissions — if the school hoped to keep that security blanket of federal money wrapped around it, that is. It seems to us a desirable result though we realize the idea will be dismissed as WASPish in some quarters. But, while any judgmental process is inherently discriminatory, a quota system imposes a degree of inflexibility on the process which diminishes the value of judgment. The quota system is, of course, easy to apply but if it assures the acceptance of a given number of units of a given qualification, it also limits the number with the same qualification which the institution must accommodate. As it imposes upon the institution a contrived obligation favoring a given group, it likewise buffers it against any obligation to that group's worthy candidates once the quota is met.

The conflicting attitudes about the decision revolve around the question of whether the principle of affirmative action has been sufficiently established that motivation to elevate minority status will continue without the coercion of quota. Minority spokesmen think not and will undoubtedly exploit the inevitable minority rejections as proof. The administrators insist their commitment is secure and will undoubtedly exploit every minority acceptance as *their* proof.

As it happens, the connection of the Bakke case with the medical world is quite incidental. The fact that the principal can now study medicine says no more for medical education or service than it does for any other academic discipline or, by extension, any other social application. Perhaps some interest attaches to the fact that a successful minority applicant, free or quota, would become a minority physician. The particular contribution of that physician to the medical care of that minority (that is, the net contribution over that which would be provided by a majority physician to that same minority) is not so much the issue as the achievement of a state of parity with the majority group in all considerations and the propagation of that state into the future. But as the quota system has relied on a quantitative treatment of the problem, its elimination enhances the qualitative value of the individual by increasing the scrutiny which must be applied to all candidates. It imposes on admissions an increased obligation to give even greater substance to the racial — or other minority-identifying — factor. In that gray zone of decision where applicants are distinguished by lesser degrees

of difference and the selection process is consequently more difficult, these minority distinctions acquire an impact not as effective in the numerical world of the quota system.

If it really works that way, the minorities respond with bitter skepticism. Well, no one can say for sure or guarantee total compliance in all areas. But the publicity attending the case has reached far beyond the medical (or judicial) arena and the intense concern with the principle involved seems to presage not a reversion to the earlier discriminatory policies but a general realization that the efforts toward equity — by all concerned — must continue even if cynics assure us that it owes more to federal money than any pervasive change of heart. And admittedly this qualitative aspect of the problem will be more difficult to assess for some time to come for there are still many more candidates — majority *and* minority — who would make good physicians than can be accommodated.

The rhetoric obscures the fact, however, that finally someone says, "This is the way it's going to be." The Bakke decision really expresses — and extends — the concept that this is the Age of the Referee. A decision is established by legislative decree (reached through the benefit of no small number of referee actions itself), is transmitted for execution to some bureaucratic entity which functions, in fact, as a referee by inflicting its interpretations of the decree on the populace. If the action is adverse to a sufficiently large or affluent segment of society, it is challenged by that group and a judicial referee tells us (eventually) whether we can do something or can't or go back to Square 1 and start over. While this process may occasionally resolve some individual point, the endless supply of new questions, the time involved in getting the referee's attention, and the occasional reversals of one referee by a higher one assure that our lives are in a continuing if fluctuating state of control by these overseers. This gives a tentative character to our society, uncertain whether our rules of today are what we think they are and if they will be the same tomorrow. This is why some are attracted to those political philosophies where the referees are more immediate, positive, and unhampered by any appeals.

In the past, the medical profession has been one of the benign — but prime — executors of this referee system and this may explain something about the disquietude it is feeling at the moment. The traditional practice of medicine (synonymous in the minds of the oldsters with "right," "real," "our way," and so on) was strictly a referee function — with the physician, of course, being the referee. The physician made the diagnosis according to the rule book and brooked no argument. He might listen with varying degrees of tolerance to others but his was the final word. His was the voice which set forth the management and therapy. The patient might or might not feel cured but if the physician-referee said he was, he was. If the role imposed upon the physician the loneliness of decision, his was the victory when the replays proved him right. The physician could even decide on his own if the patient was dead. The essential referee.

No more. Not that the referee system in medicine has been abolished. Rather, the physician's referee role has been increasingly challenged and displaced by a host of eager usurpers. His diagnosis is challenged if it doesn't agree with the patient's favorite health columnist. His advice must be passed through the filter of the second opinion. His choice of therapies is made from those available to him as a result of decisions of various referees that the method or agent is appropriate, if it can be justified financially, if it can be guaranteed to do more good than harm after consideration by a variety of nonhuman referees, and if it meets with the approval of various fiscal referees. And, of course, nothing exemplifies the intrusion of this extrinsic control as much as does that increasingly visible referee, the court.

So now, as a result of the decision of referees of high order, Mr. Bakke finds himself at the door of medical school. If he chooses to enter, his journey should at least have prepared him for the world in which he will practice. May his gratitude to the referees who made it possible sustain him as their progeny direct him in his efforts. — D.E.G.

ANNUAL MEETING

The Kansas City Society of Ophthalmology and Otolaryngology will hold its Annual Clinical Meeting on November 30-December 1, 1978 at the Hilton Plaza Inn in Kansas City, Missouri.

This Continuing Medical Education offering meets the criteria for 12 hours of credit in Category for the Physician's Recognition Award of the American Medical Association.

For reservations or additional information, write Mario J. Guastello, M.D., President, or Mr. Roger A. Weis, Executive Director, 3036 Gillham Road Kansas City, MO 64108. Telephone: (816) 531 8432.

AMA House of Delegates

Summary of Actions Taken at the 127th Annual Convention, St. Louis, Missouri, June 18-22, 1978

THIS PARTICULAR CONVENTION was extraordinary for several reasons:

Karl A. Menninger, M.D., Topeka, was the 1978 recipient of the Dr. Rodman E. Sheen and Thomas G. Sheen Award. This award is presented annually to a United States physician for scientific accomplishment. It consists of a $10,000 stipend and a plaque. The Awards Committee of the AMA Board of Trustees selects the candidate and submits the nominee to the Guarantee Bank and Trust Company of Atlantic City, New Jersey, trustee for the Thomas G. Sheen estate.

For St. Louis, the House had more than 300 recommendations, reports and resolutions with which to deal in five days. This is by far more business than had been presented to any previous meeting.

For the first time, specialty organizations were represented on a direct basis in the House. The House has frequently been called the "Congress of Medicine," and now, with fifty-five states and territories, and more than fifty specialty societies having voting representation, that description has become much more appropriate.

Before leaving this part, I just want to again emphasize that the House of Delegates is a unique deliberative body in medicine. Very few national medical groups have a policy making mechanism that allows the direct involvement of the individual physician and that deals with the total scope of medicine.

If a theme could be attached to this meeting, it would be the "cost of medical care." The House took action on responses for the recommendations of the National Commission on the Cost of Medical Care, responded to a variety of resolutions related to cost, and heard the incoming President of the Association, Dr. Tom E. Nesbitt, call for physician fee restraint.

I want to deal individually with as many of the key House actions as possible, but let me first touch on Dr. Nesbitt's inaugural address. Tom, who is a urologist in private practice in Nashville, asked each of us to restrain the rate of growth of physician fees by one per cent each year over the next two years. This doesn't mean that we should reduce existing fees across the board, and it doesn't ignore the fact that the physicians are subjected to all of the inflating pressures that affect anyone or groups of individuals who are using what amounts to a small business. It means that we should perhaps look at an increase of one per cent less than planned the next time that we review fees in terms of their relation to our costs.

This call for physician fee restraint is consistent with the AMA's involvement with the American Hospital Association and the Federation of American Hospitals in the "Voluntary Effort" program. The VE activity is designed to achieve an annual two per cent reduction in the growth of hospital costs over the next two years. Organized nationally in November 1977, and now being activated in each state, the VE is already having an impact, as the rate of growth in hospital costs already appears to have declined by more than two per cent in the first few months of this year.

Other major cost discussions centered about the 48 recommendations presented by the National Commission on the Cost of Medical Care. After a flurry of discussion, the House agreed to have the Board of Trustees and the AMA Councils study the report and submit responses to each recommendation for consideration in June. We received a 122-page report incorporating comments from the Board, the AMA Councils and three special sections about two weeks prior to the opening of the House meeting and went to St. Louis prepared to wrestle with this material.

The Commission's recommendations were its own and could not be changed. We voted on the responses to each recommendation that had been submitted by the Board. To summarize an immense volume of material, here briefly is what we did:

The House endorsed a statement that "The House of Delegates believes that the one hope for cost containment in the provision of health care lies in strengthening price consciousness in the health care marketplace."

The Commission's initial recommendations dealt with consumer price consciousness, and the House endorsed them. The principles covered in these recommendations included giving employees a periodic informed choice among various health care coverage options, giving employees economic incentives to shop for cost-effective health care coverage, and

providing for employee participation in cost decisions through co-insurance and deductibles.

Related actions endorsed providing consumers with easily understood written information about competing health care plans, certification of insurers at the state level, providing fair market competition and evaluation for provider and insurance systems, including HMOs, individual practice arrangements, and other options.

A number of Commission recommendations dealt with the assessment of quality in medical care and the use of standards and guidelines for care. In general, the House asked that the Board of Trustees and the Councils look again at this area and submit recommendations for consideration in the future. The measurement of quality care has been a difficult and elusive issue in medicine, and the Board's studies may give us some clue as to whether we can accomplish this measurement in a method satisfactory to both physicians and patients.

Aside from an endorsement of a recommendation that supported the Voluntary Effort program, the House also endorsed Commission recommendations urging experimentation with prospectively determined rates and other payment systems that can make the administration of health facilities more cost conscious. The House also endorsed the use of voluntary programs and incentives to limit bed capacity to the needs of the population served by individual hospitals and the use of experimental programs to decertify facilities that appear to be unnecessary.

The matter of public utility regulation was addressed by several recommendations. In general, the House asked for a moratorium on new regulations in the health area and a thorough evaluation of existing regulations. One of the Commission's recommendations emphasized the economic impact of regulations, a fact of life that is gaining increased attention in areas other than medicine.

Two recommendations calling for standards for the placement of expensive technical equipment and for the use of regional centers for high-cost specialized technologies were referred to the Board for study and report in December. Both are products of our time.

The question of the adequacy of physician supply was addressed by several recommendations, and the House agreed that this entire issue deserved additional study by the Board and the Councils. We have moved rapidly from a perceived physician shortage to a period wherein we have exceeded society's and our own goals for the increased production of physicians. Given the substantial resources given over the physician training and the impact that physician distribution and supply can have in the future, the results of this research can have a major impact.

The concluding recommendations from the Commission called for the physicians to receive additional information about the cost of services, both in undergraduate and graduate training and in the hospital setting, for the rapid dissemination of information about new technologies, and for a broad new approach to patient education that is consistent with the goals of our times. Most of these were adopted by the House.

A second major issue before this session of the House was a proposed revision to the Principles of Medical Ethics. In the main, the revisions have been described as both an updating of the language to reflect modern day usage and a response to legal restrictions on the health professions that have been written into state laws over the years. Some delegates expressed concern over the interpretation that could be given some portions of the revised Principles, and the House very wisely voted to refer the revisions to an ad hoc committee of the House for study and report in December. While this referral delays enactment of the revisions by a year, the importance of the Principles to all physicians justifies their careful study.

National health insurance, a subject that has traditionally received substantial attention at recent meetings of the House, was again a major topic. Five resolutions dealing with NHI were referred to the Board of Trustees with the request that any AMA sponsored NHI proposal to be introduced in the Congress next year be circulated to the House in advance of the 1978 Interim Meeting. The AMA's current proposal in Congress, with 53 sponsors, will die at the end of the year if not enacted, and it does not appear that Congress is in a mood to deal with the NHI issue in the face of substantial costs and a rising Federal deficit.

In listing priorities, I also want to touch on two other actions that relate to membership—the life blood of organized medicine. The House voted to offer membership at a 50 per cent dues level to those physicians who are in their first year of practice, an action that recognizes the economic difficulties that many physicians face in getting established after their residency training is completed.

The House also approved the reorganization of the Student Business Section to provide for a representative assembly composed of voting delegates from each of our nation's medical schools. This new structure is modeled after the AMA's Resident Physician's Section, which was restructured to provide representation from each state a year ago.

This year's meeting also marked an end to a tradition that extended back many years. The large clinical meeting that has been held in June has now been discontinued in favor of an expanded number of regional seminar programs. Research studies indicated that the regional programs better suited membership needs than the larger meeting. This kind of change is an example of the AMA's willingness and desire to change its programs and priorities to meet current needs and conserve resources.

CLAIR C. CONARD, M.D., AMA Delegate
ALEX SCOTT, M.D., AMA Delegate
LEW W. PURINTON, M.D., AMA Alternate
KERMIT G. WEDEL, M.D., AMA Alternate

Swan-Ganz

(Continued from page 493)

Abbreviations, Definitions

MAP	Mean Arterial Pressure
CO	Cardiac Output
CI	Cardiac Index
BSA	Body Surface Area
CVP	Central Venous Pressure
SVR	Systemic Vascular Resistance
PVR	Pulmonary Vascular Resistance
$\overline{PA}$	Mean Pulmonary Artery Pressure
PCW	Pulmonary Capillary Wedge Pressure
SV	Stroke Volume
SI	Stroke Index
RVSW	Right Ventricular Stroke Work
LVSW	Left Ventricular Stroke Work
P_AO_2	Partial Pressure of Oxygen in Arterial Blood
S-G	Swan-Ganz
PAO_2	Partial Pressure of Oxygen in the Alveolus (mean)
$PACO_2$	Partial Pressure of Carbon Dioxide in the Alveolus
$\bar{P}vO_2$	Partial Pressure of Oxygen in Mixed Venous Blood
$\bar{S}vO_2$	% Oxyhemoglobin Saturation of Mixed Venous Blood
CaO_2	Arterial Oxygen Content
$C\bar{v}O_2$	Mixed Venous Oxygen Content
$\dot{V}O_2$	Oxygen Consumption (STPD)
CaO_2	Mixed Venous Oxygen Content
CcO_2	Pulmonary Capillary Oxygen Content (assumed to be that content which the arterial blood would have had if fully equilibrated with mean alveolar gas)
$\dot{Q}s$	Fraction of pulmonary blood unexposed to alveolar membranes
$\dot{Q}t$	Total pulmonary blood flow
PEEP	Positive end-expiratory pressure
$\dot{Q}s/Qt$	Shunt Fraction

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KMS Obituaries

GEORGE LEE ROY ASHLEY, M.D.

Dr. George "Ed" Ashley, 65, died May 6, 1978, in Chanute.

Dr. Ashley was graduated from University of Kansas School of Medicine in 1937. He served with the U. S. Army Medical Services during World War II.

Survivors include his wife and three daughters. Memorials have been established with Neosho Memorial Hospital and St. Patrick's Church, both in Chanute.

MARGARET CLARK, M.D.

Dr. Margaret Clark, 67, died July 14, 1978, in Lawrence. She was born November 6, 1910, at Lenexa.

Dr. Clark was graduated from University of Kansas School of Medicine in 1944, and had practiced in Lawrence for more than 30 years prior to her recent retirement.

Survivors include a son. Memorial contributions may be made to the Dr. Margaret Clark Memorial Fund in care of the Lawrence Memorial Hospital Endowment Association.

SHIRLEY E. CLARK, M.D.

Dr. Shirley E. Clark, 63, died July 23, 1978, in Topeka. He was born September 18, 1914, at Rush Center.

Dr. Clark was graduated from University of Kansas School of Medicine in 1942, and served an internship in California. He served with the armed services during World War II and the Korean Conflict.

Survivors include three daughters and two sons.

JOHN L. GROVE, M.D.

Dr. John L. Grove, 97, died February 10, 1978, in Newton. He was born October 13, 1880, in Newton.

Dr. Grove served with the U. S. Army Medical Corps during World War I, and was a member of numerous civic and professional organizations.

Survivors include his wife and one son. A memorial has been established for the Axtell Christian Hospital, Newton.

MARION J. RENNER, M.D.

Dr. M. J. Renner, 82, died July 8, 1978, in Goodland. He was born April 11, 1896, in Vinland.

Dr. Renner was graduated from University of Kansas School of Medicine in 1922, and served an internship in Cleveland and residencies there and at the Hertzler Clinic prior to establishing his practice in Goodland. Known as the "Flying Doctor," he was instrumental in construction of the Goodland municipal airport, named Renner Field in his honor.

Survivors include his wife and a daughter. Memorial contributions in his name to Multiple Sclerosis may be made at Goodland State Bank or First National Bank in Goodland.



Personalities —IN KANSAS MEDICINE

William E. Ruth, Kansas City, was a participant in a recent conference on Beta$_2$ Adrenergic Agents. The conference was sponsored by Boehringer Ingelheim Ltd. and the American College of Chest Physicians, and was held in Scottsdale, AZ.

Norman C. Bos, Hutchinson, was elected secretary-treasurer of the Mid-Central States Orthopaedic Society, Inc., at their annual meeting.

A. W. Beahm, Great Bend, recently addressed students attending a course on babysitting.

Barbara Lukert, Kansas City, president of the Wyandotte County Medical Society, has been named the first recipient of the Mary F. Roberts Distinguished Professor of Nutrition.

Ernie J. Chaney, Belleville, was recently elected to the Board of Directors of the Congress of Delegates, the governing body of the American Academy of Family Physicians.

Chien Liu, Kansas City, participated in planning and served as a panelist at the 22nd Annual Symposium on Infectious Diseases of the American Association of Family Practice.

Dean Chaffee recently retired after practicing in Abilene for 32 years.

John Lynch, Topeka, discussed surgical treatment of arthritis in the final lecture of a series on "Arthritis and You" sponsored by the Arthritis Foundation with the cooperation of the Pilot Club.

Lt. Gen. Charles Pixley, surgeon general of the U. S. Army, recently presented the Major Louis Livingston Seaman Award to **Robert E. Miller,** Garden City.

Jimmie A. Gleason, Topeka, spoke recently on teen-age pregnancies at a Parent-Teacher Association meeting.

Lee S. Fent, Newton, addressed the annual dinner meeting of the Crawford County Unit of the American Cancer Society. His topic was "Cancer—Where We Are Today."

Tyrone Artz, Wichita, presented a program on hand surgery at a recent meeting of the Butler-Greenwood County Medical Society.

Neil Schimke, Kansas City, was lecturer for the recent Third Annual Harper Seminar Series on the subject of pre- and post-natal genetics.

A. B. McConnell was honored at a recent reception following his retirement from nearly 58 years of practice in the Burlington community.

Geoffrey Logan, Wichita, spoke on "Indications for Cesarean Birth" at a recent meeting of the Cesarean Delivery Support Group.

Karl Kavel, Topeka, recently addressed the Greater Topeka Better Breathing Club on the topic "Allergies and Breathing."

Leo Cawley, Wichita, was featured speaker during the recent annual meeting of the Kansas Chapter, Arthritis Foundation. **Dennis Finley,** Wichita, was elected president, and **Richard Lies,** Wichita, was elected to the Governing Board.

Benjamin M. Matassarin, Wichita, has been appointed to the state Advisory Commission on Institutional Management and Community Mental Health Programs.

Donald D. Goering, Salina, participated in a discussion on "Pastoral and Medical Intervention in Drug Related Problems" at a workshop sponsored by the Pastoral Care Advisory Committee.

Andrew Nachtigall, Newton, spoke on "The Medical Aspects of Babysitting" at a recent babysitting clinic.

R. B. White, Winfield, was a lecturer at a recent medical seminar in Harper for health professionals from nine counties.

Arthur Cherry, Topeka, addressed a group of students at Topeka High School on what is happening in family-infant interaction. His talk was part of a mini-course on child abuse.

Max E. Teare, Garden City, was featured speaker at the University of Missouri Department of Psychiatry graduation banquet. His topic was "Rural Mental Health Care and Its Delivery System."

Jess Koons, Liberal, spoke on "How Children See," at a recent meeting of the Southwest Kansas Medical Assistants.

A. L. Tocker, Sulenan Sadig and **Krishan Goyle,** all of Wichita, presented a program on Echo-cardiography at a recent meeting of the Butler-Greenwood County Medical Society.

Albert P. Michelbach, Goddard, has received the 1978 Outstanding Volunteer Award for service to the Sedgwick County Heart Division and to the Kansas Affiliate.

At Fellowship Meals at Douglass Center, **George S. Bascom,** Manhattan, discussed health problems that confront the elderly.

Robert H. O'Neil, Topeka, has been appointed to the Kansas Board of Regents.

Thomas Dechairo, Westmoreland, addressed the Governor's Conference on Aging on the Topic of Intimacy: Intellectual Openness, Emotional Access, and Sexual Availability.

Vox Dox

Vox Dox Editor:

I recommend that physicians pay some attention to the manner in which pharmacists are filling prescriptions. I have found that a large number of pharmacies will fill prescriptions for smaller amounts than are ordered in order to get the patient to come back and have multiple refills, each one of course with the "professional service" component. Thus, a prescription written for 100 pills may well be filled for 30 or 40, three times. Since most patients don't know how to read prescriptions, they just assume the pharmacist is doing as the physician instructed. Since the "professional service" component may be anywhere from $1 to $5 per refill, this can turn into a very significant increase in cost. It is particularly significant when patients are on maintenance doses of medication for chronic diseases such as epilepsy, gout, hypertension, etc. You would not find out about this except by questioning your patients. But don't be surprised if you wrote a prescription for 100 pills with three refills for 100 each that the patient ended up going to the pharmacist 16 times rather than four. There seems to be no reliable defense against this, but I am going to try to save my patients money by having my next batch of prescriptions pads printed "Refill for exactly __ at one time." You might wish to consider the same.

James W. Wiggs, M.D.
1027 Jackson
Great Bend, KS 67530

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Kansas Press Looks at Medicine

When local physician Dr. Alex Scott attended the American Medical Association annual convention in late June in St. Louis as a Kansas delegate, he was interviewed by Richard A. Knox, reporter from *The Boston Globe*. Knox wired the interview as a special to *The Washington Post*.

Dr. Scott, pleased at the accuracy of the article, wrote a letter to *The Boston Globe* in appreciation. In part he noted, "He (Knox) interviewed me just after the AMA President's speech in a jostling crowd where there were many distractions. The article which he wrote, and which I saw in *The Washington Post*, was absolutely accurate with the quotations verbatim. He correctly interpreted my comments and this was done with very little opportunity for good note-taking. I was impressed by his excellent auditory memory. Sometimes I think the Fourth Estate catches as many barbs and arrows as we do in medicine. For that reason, I want you to know that one small town Kansas doctor does appreciate you."

The headline on that letter was "Imprimatur" (let it be printed), indication of a sort of "Hallelujah" reaction from the press which gets complaints more often than compliments.

One thing leads to another. Dr. Scott received a surprised note from a pharmacy sales representative who had moved from Kansas City to Boston. Friend and grateful patient who had some nice things to say about a "small town Kansas doctor."

Dr. Scott in *The Boston Globe* and a random sprinkling from the *Junction City Daily Union* in the Ft. Lauderdale daily where Johnny Wolin holds down the sports desk . . . those made our week. Junction City has some good things going for it for a change.—*Junction City Daily Union*, Aug. 1, 1978.

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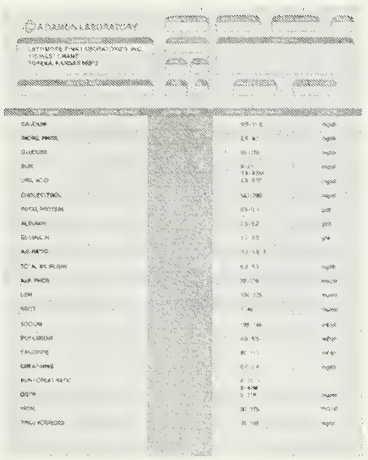
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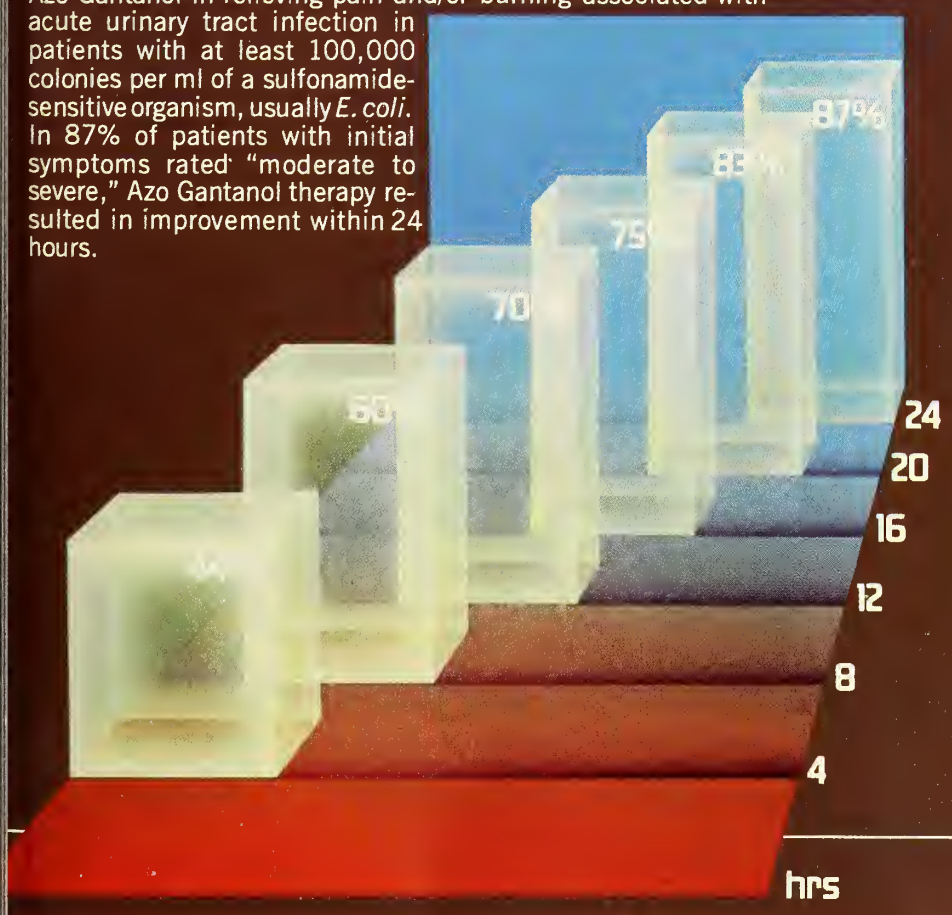
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Contraindications: Children below age 12; sulfonamide hypersensitivity; pregnancy at term and during nursing period; because Azo Gantanol contains phenazopyridine hydrochloride it is contraindicated in glomerulonephritis, severe hepatitis, uremia, and pyelonephritis of pregnancy with G.I. disturbances.

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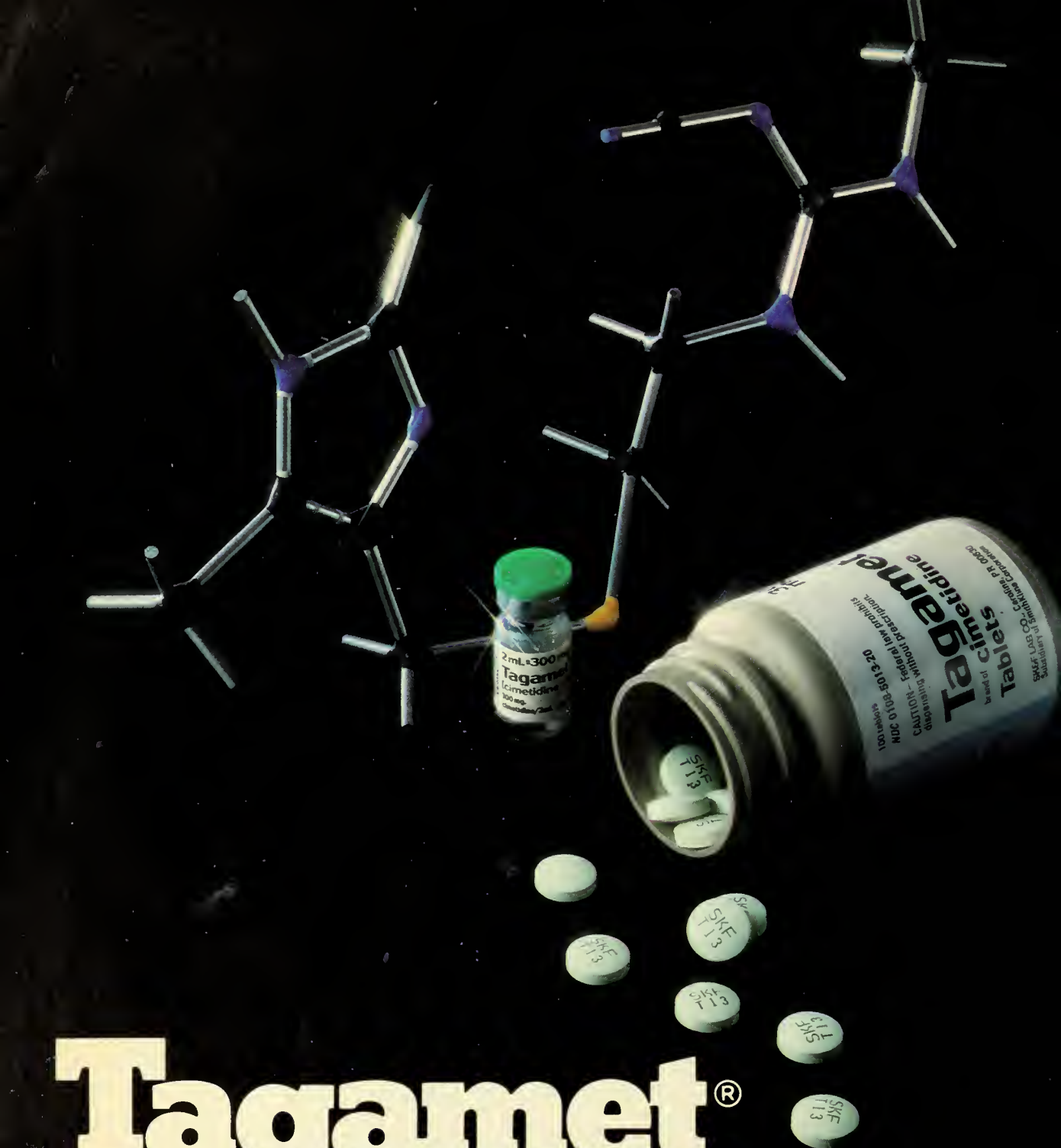
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Each tablet contains: aspirin, 227 mg; phenacetin, 162 mg; and caffeine, 32 mg; plus codeine phosphate in one of the following strengths: #4—60 mg (gr 1); #3—30 mg (gr ½); #2—15 mg (gr ¼); and #1—7.5 mg (gr ⅛). (Warning—may be habit forming). C III

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For recurrent attacks of urinary tract infection in women

Bactrim™ DS Double Strength Tablets

Each tablet contains 160 mg trimethoprim and 800 mg sulfamethoxazole.



Just one tablet b.i.d. for 10 to 14 days

- Action at urinary/vaginal/lower bowel sites helps eliminate reservoirs of infecting organisms
- Distinctive antibacterial action plus wide spectrum helps eradicate recurrent UTI
- Low incidence of bacterial resistance in community practice
- Convenient *b.i.d.* dosage provides day-and-night antibacterial control
- Contraindicated during pregnancy and the nursing period. During therapy, maintain adequate fluid intake; perform CBC's and urinalyses with microscopic examination.

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* **It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination.** *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

The recommended quantitative disc susceptibility method (*Federal Register,* 37:20527-20529, 1972) may be used to estimate bacterial susceptibility to Bactrim. A laboratory report of "Susceptible to trimethoprim-sulfamethoxazole" indicates an infection likely to respond to Bactrim therapy. If infection is confined to the urine, "Intermediate susceptibility" also indicates a likely response. "Resistant" indicates that response is unlikely.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older:

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension,* containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

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Please see back cover.

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Her next attack of cystitis may require the Bactrim™ 3-system counteratta



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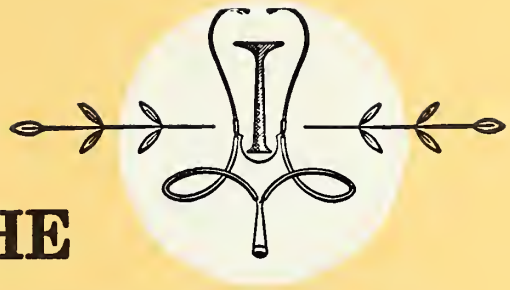
Bactrim has shown high clinical effectiveness in recurrent cystitis as a result of its wide spectrum and distinctive antimicrobial action in the urinary, vaginal and lower intestinal tracts.

The probability of recurrent urinary tract infection appears to be enhanced by the establishment of large numbers of *E. coli* or other urinary pathogens on the vaginal introitus. The trimethoprim component of Bactrim diffuses into vaginal fluid in effective concentrations, thus combating migration of pathogens into the urethra.

Studies have shown that Bactrim acts against *Entero bacteriaceae* in the bowel without the emergence of resistant organisms. Thus, Bactrim reduces the risk of introital colonization by fecal uropathogens. It has *no* significant effect on other normal, necessary intestinal flora.

Bactrim fights uropathogens in the urinary tract/vaginal tract/lower intestinal tract

Please see reverse side for summary of product information.



THE Journal OF THE Kansas Medical Society

OCTOBER
1978

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NO. X

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The JOURNAL of the KANSAS MEDICAL SOCIETY

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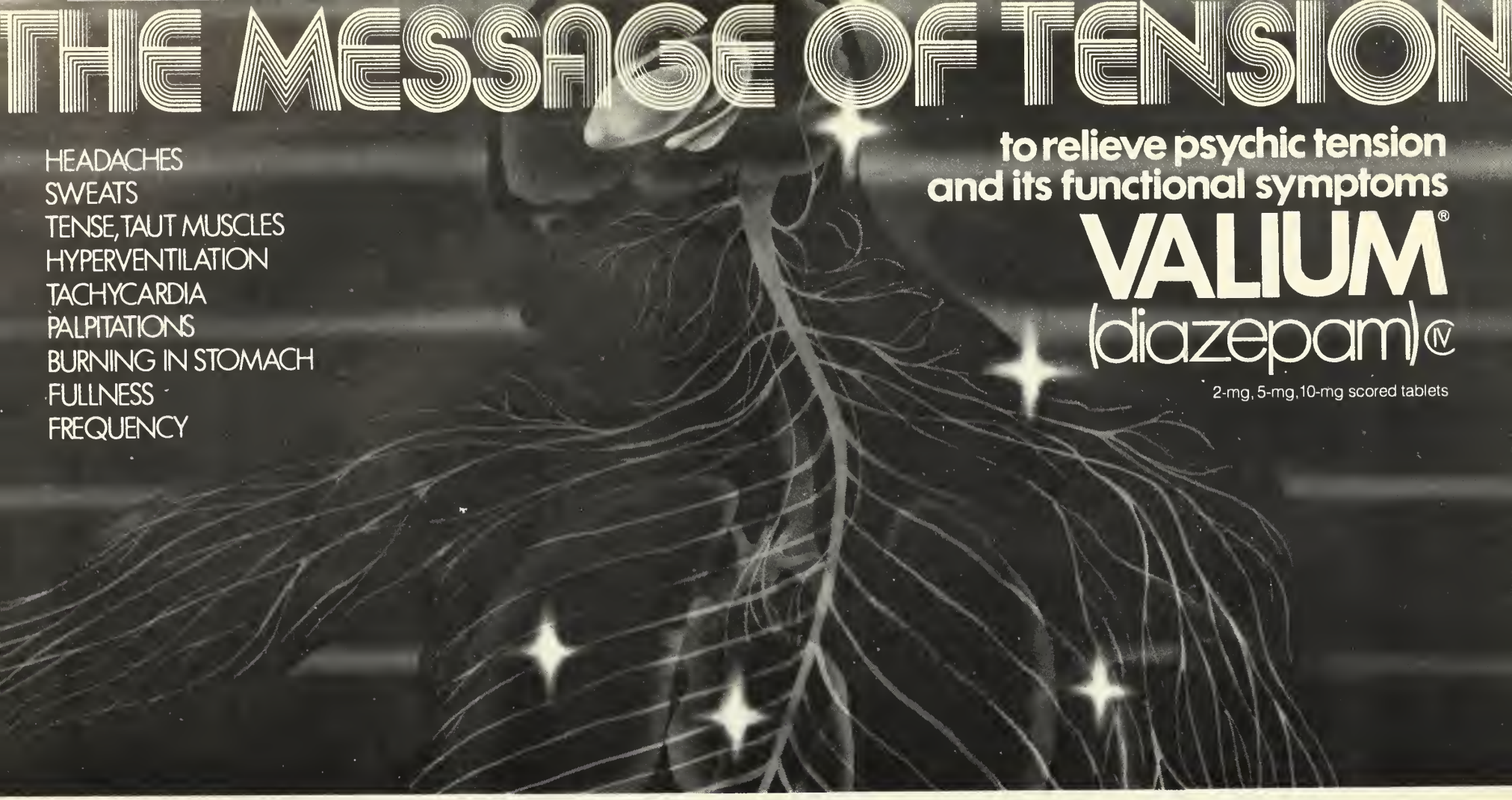
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The effectiveness of Valium in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy.
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed, drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

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Auxiliary News

Auxiliary News

October is the month for the AMA Auxiliary Fall Confluence. This is a concentrated three-day training session for auxiliary leaders. State presidents, presidents-elect, and a certain number of county presidents-elect are invited to attend. Kansas is allowed four county presidents-elect, based on our total membership.

Many good auxiliary programs have been curtailed or dropped completely because a decrease in membership has made it financially impossible to continue them. I am grateful that the Fall Confluence is not one of the discontinued programs. The seminars and workshops offered are of top quality and are immeasurably helpful to those who are leading or preparing to lead their own auxiliaries.

I strongly urge each of you to consider your relationship to the AMA, and that of your spouse to the AMA Auxiliary. I realize that we may not always agree with all the policies and decisions of the respective organizations. However, these are the organizations that can speak for us and we certainly can have no voice in policy-making decisions unless we are members. My personal belief is that the pros far outweigh the cons and that membership is a privilege.

Thank you for your continued support. Without you we could not exist. Again, may I renew our pledge to assist and support you in whatever way you may deem advisable.

Sincerely,
Jean Crouch
President
Kansas Medical Society Auxiliary

Tenuate®
(diethylpropion hydrochloride NF)

Tenuate Dospan®
(diethylpropion hydrochloride NF) controlled-release

AVAILABLE ONLY ON PRESCRIPTION
Brief Summary
INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.
CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).
WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle, the patient should therefore be cautioned accordingly. *Drug Dependence*. Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*: Tenuate is not recommended for use in children under 12 years of age.
PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.
ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema. *Endocrine*: Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.
DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.
OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

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References: 1. Citations available on request—Medical Research Department, MERRELL RESEARCH CENTER, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon, R.H., and Leyland, H.M. A Comprehensive Review of Diethylpropion Hydrochloride. International Symposium on Central Mechanisms of Anorectic Drugs, Florence, Italy, Jan. 20-21, 1977.

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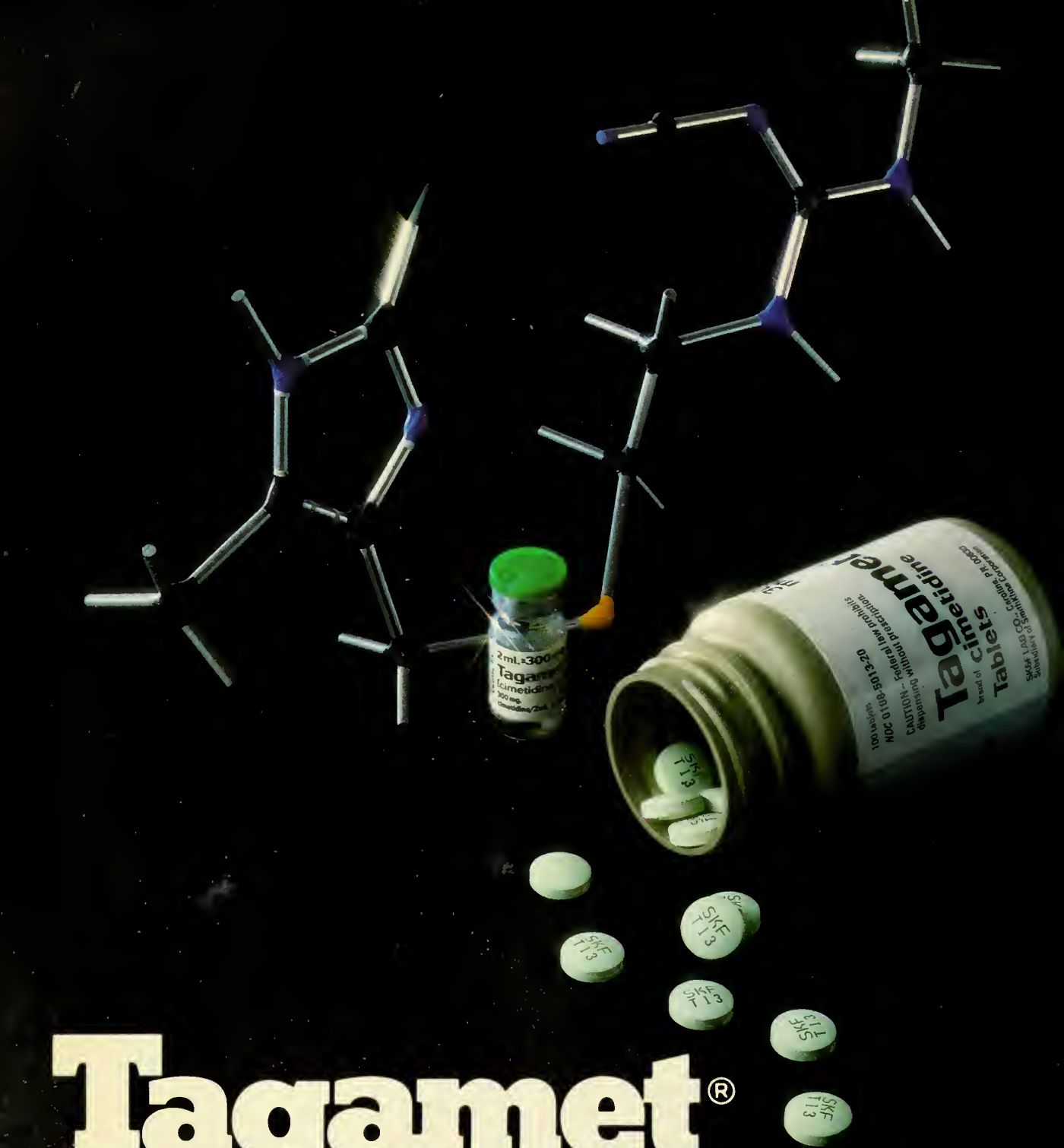
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Precautions: Use with caution in patients with cardiac disease, hepatic or renal impairment. Concurrent administration with certain antibiotics, i.e. clindamycin, erythromycin, troleandomycin, may result in higher serum levels of theophylline. Plasma prothrombin and factor V may increase, but any clinical effect is likely to be small. Metabolites of guaifenesin may contribute to increased urinary 5-hydroxyindoleacetic acid readings, when determined with nitrosonaphtol reagent. Safe use in pregnancy has not been established. Use in case of pregnancy only when clearly needed.
Adverse Reactions: Theophylline may exert some stimulating effect on the central nervous system. Its administration may cause local irritation of the gastric mucosa, with possible gastric discomfort, nausea, and vomiting. The frequency of adverse reactions is related to the serum theophylline level and is not usually a problem at serum theophylline levels below 20 μg/ml.
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ADDENDUM TO 1978 ROSTER

Any physician who has been a member of the Kansas Medical Society for ten years may, at the age of 70, apply for emeritus status. Upon approval, they then remain members of the Society without payment of dues.

The 1978 Roster erroneously listed all those physicians with emeritus status as retired, which is not necessarily the case. These physicians are listed below with their medical specialities.

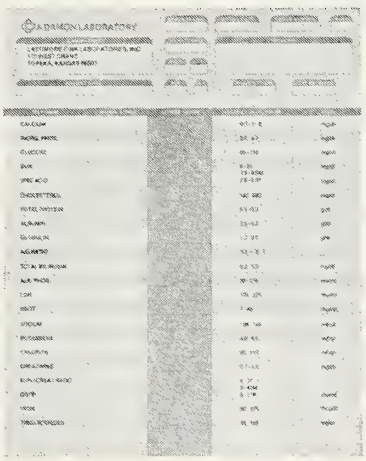
Alexander, Clyde W.	Kansas City	FP
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Ashmore, Arthur L.	Wichita	GS
Bartlett, Wayne C.	Wichita	GS
Baude, Eugene L. Andre	Topeka	IM
Beebe, Edmer	Olathe	FP
Beelman, Floyd C.	Topeka	FP
Bennett, Charles A.	Leavenworth	FP
Biermann, Aloysius H.	Garden Plain	FP
Brian, Robert M.	El Dorado	GS
Bribach, Eugene J.	Atchison	OPH
Brown, Virgil E.	Sabetha	FP
Bryant, Homer L.	Coffeyville	OPH
Bullock, Harold O.	Independence	IM
Butcher, Thomas P.	Emporia	GS
Chalian, Alexander R.	Kansas City	A
Cooper, Arthur E.	Norton	FP
Davis, David R.	Emporia	PD
Delp, Mahlon H.	Shawnee Mission	IM
Dlabal, Frank A.	Wilson	FP
Dyck, Arthur H.	McPherson	FP
Elnen, Walter T.	Wichita	GS
Enders, Wray	Shawnee Mission	A
Evans, Farris D.	Wichita	FP
Gelvin, E. Raymond	Concordia	GS
Gerber, Harry A.	Leavenworth	FP
Gilbert, J. Howard	Seneca	FP
Gollier, Robert A.	Ottawa	FP
Gradinger, Billens C.	Halstead	IM
Gsell, George F.	Wichita	OPH
Hawkey, Alfred S.	Newton	OBG
Henneberger, Charles E.	Atwood	GS
Henning, Calvin W.	Ottawa	FP
Hiebert, Peter E.	Kansas City	R
Hinshaw, Charles T.	Wichita	PD
Hodson, Hervey R.	Wichita	GS
Huston, Francis W.	Winchester	FP
Janzen, Herman F.	Hillsboro	FP
Johnson, Frederick E.	Topeka	FP
Johnson, Joseph H.	El Dorado	EENT
Kassebaum, Glen E.	El Dorado	GS
Keys, Robert C.	Winchester	ORS
Kiser, Willard J.	Wichita	GS
Kosar, Clarence D.	Concordia	EENT
Lawson, Dwight	Topeka	IM
Little, L. Gilbert	Wichita	P
Lofgreen, Victor J.	Ottawa	FP
Lowe, DeWitt S.	Hiawatha	FP
McConnell, Archie B.	Burlington	GP
Medearis, Donald N., Sr.	Kansas City	PD
Meidinger, Ray	Hiawatha	FP
Menninger, Karl A.	Topeka	P
Miller, Herbert C.	Kansas City	PD
Mills, Charles D.	Wichita	FP
Mullen, Clifford J., Sr.	Kansas City	OPH
Neighbor, Ernest G.	Shawnee Mission	FP
O'Donnell, Leonard A., Sr.	Wichita	IM
Oltman, Theodore V.	Riley	FP
Pettijohn, Walter J.	Russell	GS
Pinsker, Jacob A.	Wichita	IM
Plowman, Carl W.	Jewell	FP
Price, Vaughn C.	McPherson	GS
Pyle, Lucien R.	Topeka	OBG
Reece, Adelbert S.	Gardner	FP
Regier, Henry L.	Kansas City	GS
Relihan, Francis H.	Smith Center	FP
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Final classification of the less-than-effective indications requires further investigation.

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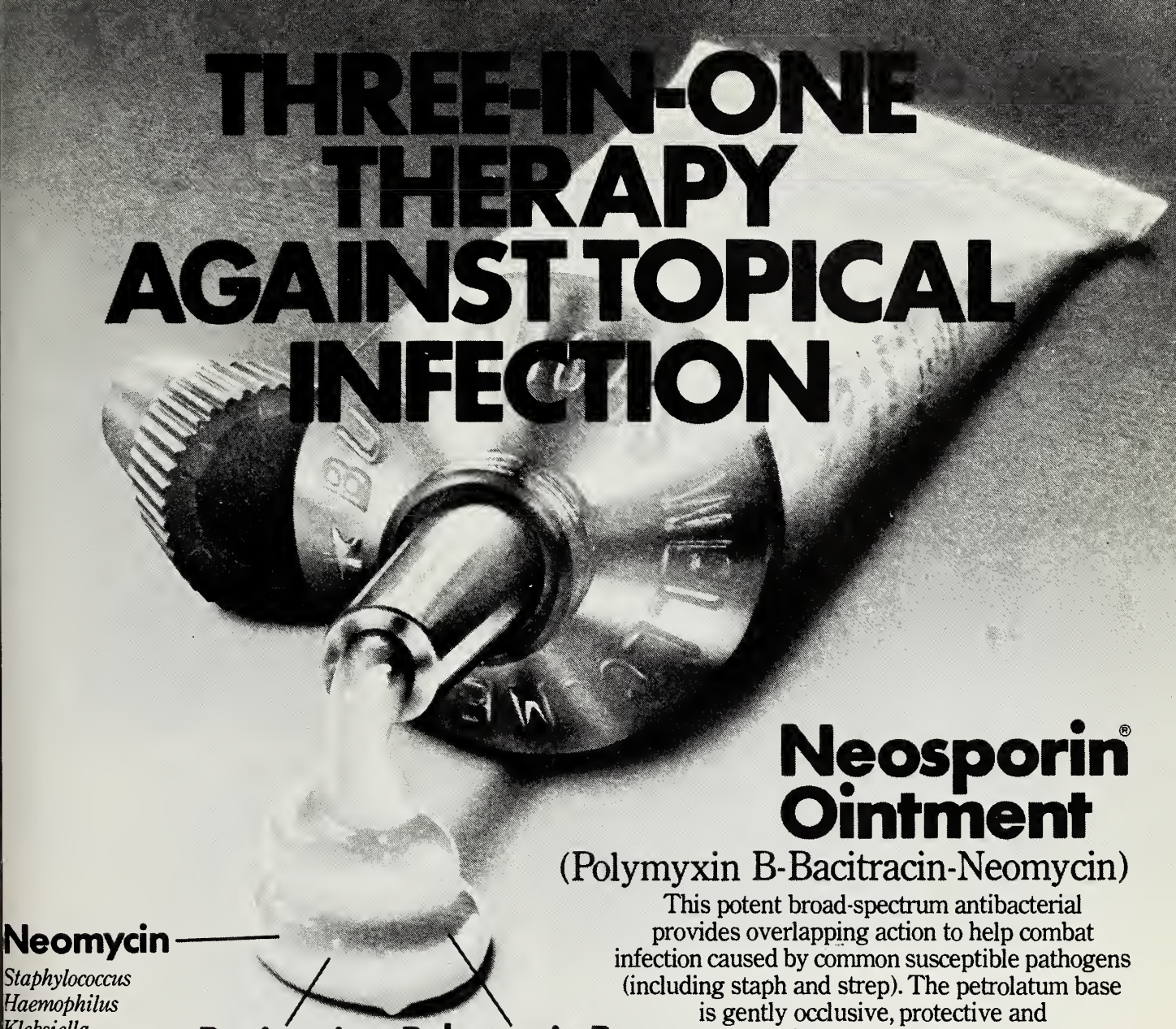


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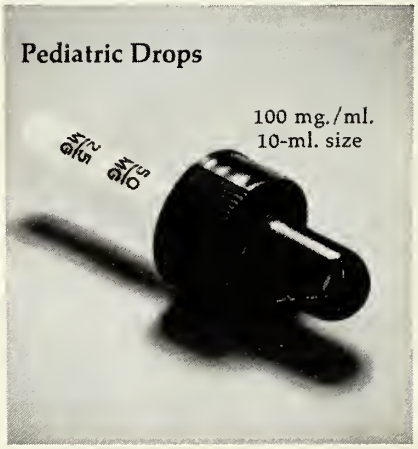
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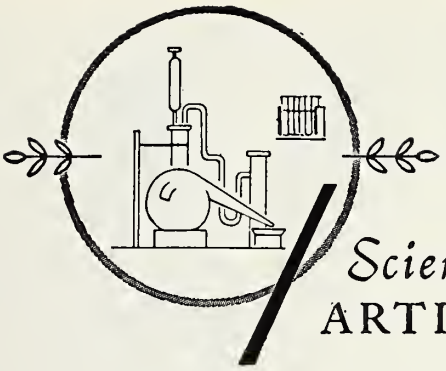


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Scimitar Syndrome

Surgical Considerations

THOMAS V. THOMAS, M.D.; MARIO L. GUMUCIO, M.D. *and* **NALINI PREMSINGH, M.D.,** *Kansas City, Kansas*

The prognosis is excellent for patients suffering from this congenital anomaly. The physiology of the defect is discussed, and a corrective surgical technique is presented.

SCIMITAR SYNDROME is a complex, congenital anomaly of the lung and heart featuring abnormal venous drainage from the lung into the inferior vena cava. Supradiaphragmatic as well as infradiaphragmatic drainage of the lung may be encountered with or without an associated inter-atrial septal defect. This is a relatively rare anomaly compared to the partial anomalous venous return frequently encountered in patients with atrial septal defects. Scimitar syndrome usually presents a single channel draining the entire right lung into the inferior vena cava, resulting in a large left-to-right shunt. This channel runs parallel to the right heart border and frequently enters the abdominal cavity through the diaphragm. Infrequently, patients with scimitar syndrome have multiple associated anomalies such as hypoplasia of the right lung, partial anomalous drainage of the right lung, systemic arterial supply to the right lower lobe, bronchial malformations, bronchiectasis, intra-cardiac defects, and diaphragmatic variations such as eventration and duplication.[1, 2] Extrathoracic congenital anomalies are uncommon in scimitar syndrome.

The first established report of this entity was made in 1949 by Dotter, *et al.*[3] The characteristic roentgenographic appearance of the scimitar was attributed to this anomalous vein by Neill and coworkers.[4] Before the advent of cardiopulmonary bypass, total correction of this anomaly was not feasible and pulmonary resection was the only attempted treatment. Hemodynamic correction was first carried out by Kirklin and associates in 1956.[5] Surgical treatment has frequently involved implantation of the anomalous vein into the left atrium, implantation into the right atrium and use of a pericardial tunnel, use of the anterior wall of the right atrium as a baffle, or creating a tunnel from the inferior vena cava into the left atrium with pericardium or Dacron patch.[1, 6-10] Repair has been accomplished with cardiopulmonary bypass alone and with cardiopulmonary bypass, hypothermia, and circulatory arrest. Point of entrance of the anomalous vein into the inferior vena cava is inaccessible during conventional technique of cardiopulmonary bypass; this poses technical problems during the repair. Implantation of the anomalous vein directly into the left

Address reprint requests to Dr. Thomas, 4631 Orville Ave., Suite 211, Kansas City, KS 66102.



Figure 1. Posterior-anterior view of the chest, showing the scimitar parallel to the right heart border. There is mild cardiomegaly.



Figure 2. A catheter is shown passed from the inferior vena cava into the anomalous vein up to the level of the upper lobe of the right lung. Contrast material was injected to opacify the vein more clearly.

atrium has usually resulted in stricture or kink and is not considered to be an acceptable approach today. Our experience with a modified technique for correction of scimitar syndrome has resulted in expediency and simplicity of repair.

Case Report

A 19-year-old white female was admitted to Bethany Medical Center in Kansas City, Kansas, in March 1977, with pain in the lower, right side of the chest, shortness of breath, and tiredness of several months' duration. Chest x-ray revealed a large paracardiac shadow highly suggestive of total anomalous venous drainage of the right lung into the inferior vena cava. Electrocardiogram showed evidence of incomplete right bundle branch block and right ventricular hypertrophy. During cardiac catherization, oxygen saturation in the inferior vena cava was found to be 87 mm Hg. The catheter was advanced into the anomalous vein without difficulty, to opacify and delineate passage of the anomalous vein into the inferior vena cava below the diaphragm.

During the surgical correction of this anomaly, mediastinotomy incision was employed. The right mediastinal pleura was opened and a large anomalous vein measuring approximately 2½ cm in diameter was encircled with umbilical tape for temporary occlusion during the repair to obtain a dry field. Ascending aorta and superior vena cava were cannulated in the usual manner for cardiopulmonary bypass. The inferior vena caval cannula, however, was inserted through the right common femoral vein. The diaphragm was divided up to the vena cava to obtain more exposure of the vena cava. This also facilitated downward retraction of the liver. Ice cold saline was poured into the pericardial cavity and anoxic cardiac arrest was obtained by cross clamping the ascending aorta. The inferior vena cava was occluded at the level of the entrance of the hepatic veins.

The incision into the right atrium was extended

along the inferior vena cava to the level of entrance of the scimitar vein. A segment of pericardium approximately 3 cm wide and 6 cm long was sutured around the entrance of the scimitar except at the superior border of the opening, and continued into the right atrium creating a tunnel for passage of the blood upwards from the inferior vena cava. A 3-cm atrial septal defect was created starting at the level of the foramen ovale, and the pericardial patch was sutured all around this defect except at the inferior border. Then the incision in the vena cava and right atrium was closed using continuous suture. The ascending aorta and the left ventricle were aspirated for residual air, and the aortic clamp was released. No vent was used for decompression of the left side of the heart.[11] The heart was defibrillated and cardiopulmonary bypass was discontinued. The total cardiopulmonary bypass time was 42 minutes, and no blood transfusions were required for the operative procedure or in the post-operative period.

This patient has made an uneventful recovery with significant improvement in her functional capacity. She married a month after dismissal from the hospital and recently gave birth to a child.

Comments

The majority of patients with scimitar syndrome remain asymptomatic until their teen or adult years. Drainage of the right pulmonary venous blood into the inferior vena cava creates the same physiological change as an atrial septal defect. Decompensation occurs early if at least 50 per cent of the pulmonary venous blood enters the right side of the heart. Pulmonary resection as a method of correction is no longer considered a treatment of choice. The anomalous vein is usually too short and thin-walled for division from the vena cava and implantation into the left atrium. Implantation into the right atrium with creation of a pericardial tunnel into the left atrium with repair of the opening into the inferior vena cava is tedious and carries the risk of being too short or getting kinked. Anomalous venous drainage into the inferior vena cava above the diaphragm can be corrected with creation of septal defect and patch repair using a right atrial incision without significant difficulty.

Infradiaphragmatic entrance of the anomalous vein into the inferior vena cava adds considerably to the technical difficulties while the patient is on cardiopulmonary bypass. Temporary interruption of cardiopulmonary bypass and removal of the inferior vena caval cannula under hypothermia of usually 20C has also been frequently used. However, by the method described above, the inferior vena caval cannula is placed from the femoral vein, and hypothermia or interruption of the cardiopulmonary bypass is not necessary. Removal of the inferior vena caval cannula from the site of creation of the tunnel, and division of the diaphragm, adds greatly to the surgical expediency and technical simplicity. Scimitar syndrome, although rare and complex, is a totally correctable anomaly with low morbidity and mortality.

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The Infected Mitral Valve

Floppy Mitral Valve Syndrome Complicated by Haemophilus Parainfluenzae Bacterial Endocarditis

JAMES E. COKER, M.D.; DAVID L. DWORZACK, M.D.; GLENN R. HODGES, M.D.; SAMUEL C. PETRIE, M.D., *and* DAVID M. PUGH, M.D., *Kansas City*

This case, complicated by *Haemophilus parainfluenzae* endocarditis, is remarkable in that embolic complications did not occur, and when seen six months after completion of therapy, the patient remained in good health. The antibiotic combination of gentamicin and ampicillin, synergistic in vitro against the patient's infecting organism, was effective in the treatment of this patient.

ALTHOUGH REID[1] focused attention to the intracardiac origin of midsystolic clicks and murmurs, the cause was not known until Barlow[2] demonstrated mitral valve regurgitation by cineangiography in eight patients with late systolic murmurs. Reid coined the term floppy mitral valve syndrome descriptive of the redundancy of the mitral valve leaflets and prolapse of the valve causing regurgitation.[1] Histologically there is myxomatous degeneration of the mitral valve apparatus characterized by mucopolysaccharide deposition disrupting the normal connective tissue.[3]

Bacterial endocarditis is a significant cause of morbidity in patients with mitral valve prolapse. The reported incidence of this complication ranges from 2-6 per cent.[4-8] This report describes a patient with mitral valve prolapse who developed *Haemophilus parinfluenzae* endocarditis. As this is an unusual cause of endocarditis in a patient with a common predisposing condition, the case is reported in detail.

Case Report

A 38-year-old male presented with a two-week history of fever, chills, night sweats, myalgias, and headache. A cardiac murmur had first been noted three years earlier. There was no history of recent dental work or operative procedures. Physical examination showed an alert, well developed male. Examination revealed temperature, 38.5C; blood pressure, 120/80 mm Hg; pulse, 90 beats/min; respiration rate, 14/min; height, 1.9 M; and weight, 72 kg. Sclerae, fundi, and conjunctivae were free of hemorrhages or petechiae. Pectus excavatum and rales at the posterior base of the left lung were noted. Cardiovascular examination revealed the point of maximal impulse and the first and second heart sounds to be normal. A midsystolic click followed by a grade II/VI systolic murmur were heard at the left sternal border. The click moved closer to the first heart sound and the murmur maintained its intensity when the patient was standing. Venous and carotid pulses were normal. Abdominal examination revealed a palpable spleen. The remainder of the physical examination showed normal results.

Admission laboratory studies produced the following values: hemaglobin, 13.8 gm/100 ml; WBC, 11,400/cu mm with 2% metamyelocytes, 15% band form polymorphonuclear leukocytes, 65% neutrophils, 12% lymphocytes, and 6% monocytes; and toxic granulation of neutrophils. Microscopic examination of urine revealed leukocytes and erythrocytes. Rhematoid factor was reactive at a titer of 1:80. The results of the electrocardiogram and chest roentgenogram were unremarkable. Four blood cultures grew *H. parainfluenzae*.

Initially, the patient was treated with ampicillin, 12 gm intravenously/day, and gentamicin, 240 mg intramuscularly/day, for five days. Ampicillin, 12 gm intravenously/day, was then continued alone for an additional seven days. An erythematous pruritic rash prompted change of antibiotic to cephalothin, 8 gm intravenously/day, for seven days.

From the Divisions of Infectious Disease and Cardiology, Department of Medicine, UKSM-Kansas City, KS and Medical Service, V. A. Hospital, Kansas City, MO.

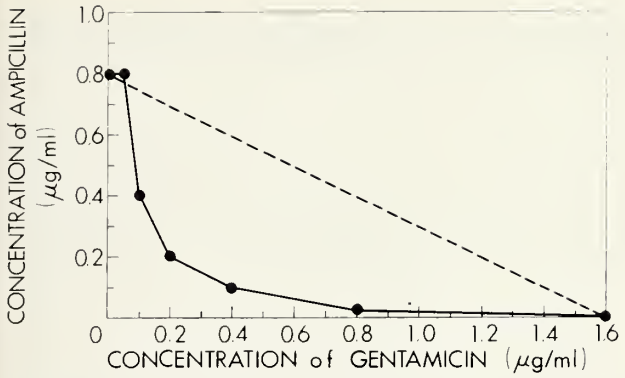


Figure 1. Isobologram. The dashed line connects the two minimum inhibitory concentrations (MIC) of ampicillin and gentamicin for the organism. The solid line bowing below the dashed line is the experimental finding demonstrating in vitro synergy.

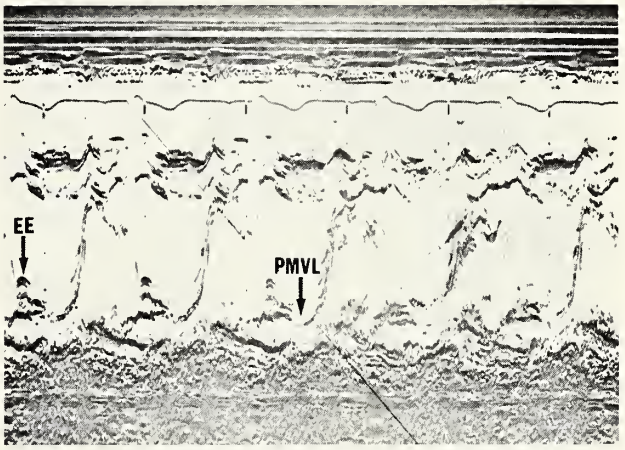


Figure 2. Initial Echocardiogram. Obtained during active phase of endocarditis, this echocardiogram demonstrates extra echoes during systole on the mitral valve and prolapse of the mitral valve posterior leaflet during systole. EE = extra echoes, PMVL = prolapse mitral valve leaflet.

Due to presumed penicillin allergy, persistent fever and night sweats on therapy, the patient was transferred to the University of Kansas Medical Center. Therapy with ampicillin was reinstituted and the dose titrated up to 12 gm intravenously/day. Gentamicin, 225 mg intravenously/day, was added after in vitro synergy between ampicillin and gentamicin against the patient's *H. parainfluenzae* was demonstrated *(Figure 1)*. The combination of ampicillin and gentamicin was administered for 15 days with resultant lysis of fever and cessation of night sweats. Serum bactericidal titers $\geq$ 1:16 at the anticipated nadir of antibiotic concentrations were maintained during this time.

Documentation of the mitral valve prolapse was obtained by echocardiography. In addition, the initial echocardiogram demonstrated multiple echoes suggestive of mitral valve vegetation *(Figure 2)*.

The patient is still well six months after hospitalization. Blood cultures remain sterile and a repeat echocardiogram confirmed mitral valve prolapse and absence of the previously noted "extra echoes" *(Figure 3)*.

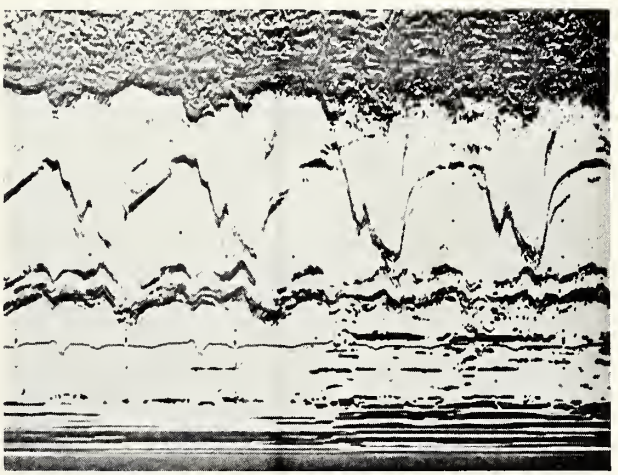


Figure 3: Follow-up echocardiogram. Diminuition of the initially noted extra echoes of the mitral valve during systole are demonstrated.

Comment

Our patient fits into the subset of patients with mitral valve prolapse and a late systolic murmur who are more prone to complications than patients with only a midsytolic click.[5] The complications encountered in mitral valve prolapse are ventricular arrhythmia, progressive mitral insufficiency, and bacterial endocarditis.[4-8]

The occurrence of bacterial endocarditis in the mitral valve prolapse syndrome ranges from 2-6 per cent.[5, 6] In a recent review of bacterial endocarditis, 10 of 87 cases occurred in patients with mitral valve prolapse syndrome existing before infection.[4] In the same review, of the 28 cases of endocarditis associated with mitral regurgitation, 10 were due to mitral valve prolapse syndrome. Bacterial endocarditis in patients with mitral valve prolapse is complicated acutely by cerebral emboli in 25 per cent, and progressive congestive heart failure due to mitral valvular insufficiency in 24 per cent.[4]

In two recent reviews,[9-10] the incidence of *H. parainfluenzae* as a causative organism in 577 cases of endocarditis was less than 0.9 per cent. Even in the pediatric age group where *Haemophilus* species

infections are more common, no cases of *H. parainfluenzae* endocarditis were found in one review.[11] In the last twenty years, there have been 17 reported cases of *H. parainfluenzae* infective endocarditis.[5, 12-14] To our knowledge, only one other case of *H. parainfluenzae* endocarditis superimposed upon mitral valve prolapse syndrome has been reported.[5]

Patients reported with *H. parainfluenzae* endocarditis have a predisposition to embolic occlusion of major arteries in 60-80 per cent of reported cases.[5, 12-14] This incidence of arterial emboli is presumably due to large friable vegetations similar to those of fungal endocarditis.[12, 13] Arterial emboli were not observed in the case reported.

Echocardiography has been documented as a sensitive method for detection of both mitral valve prolapse and valvular vegetations.[15, 16] The initial echocardiograms from our patient showed prolapse of the posterior leaflet of the mitral valve and extra echoes, suggesting valvular vegetations. The follow-up echocardiogram revealed no extra echoes but persistent prolapse. Recently, the value of echocardiography in showing valvular vegetations in patients with mitral valve prolapse and endocarditis has been questioned.[17] Thus, the meaning of the disappearing echoes in our patient's echocardiograms is uncertain.

Recent reports have suggested that ampicillin combined with gentamicin is the regimen of choice for treating *H. parainfluenzae* endocarditis.[12, 13] After the combined activity of gentamicin and ampicillin were shown to be synergistic using the procedure described by Sabath,[18] this regimen was employed with apparent cure of the infection in our patient.

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PATIENT PACKAGE INSERTS

Currently, the Food and Drug Administration (FDA) requires patient information to be distributed with estrogens, oral contraceptives and isoproterenol inhalation products. In the near future, FDA shall announce its intent to eventually require Patient Package Inserts (PPIs) for a wide range of drug products on the market. The first group of PPIs shall be drafted under contract. FDA shall ask bidders to draft prototype PPIs for a few selected drugs.

In order to gather advice from a variety of perspectives regarding the contents and methods of presentation for PPIs, FDA is sponsoring a Conference December 11-12, 1978, at the Shoreham Americana Hotel in Washington, D.C.

The theme for this Conference will be focused specifically on how to choose contents and methods of presentation for selected prototype drugs. Input from health professionals, consumers and other interested parties is solicited.

Registration is free, but in order to arrange workshops, preregistration is heavily encouraged. Requests for information and registration forms should be directed to: Ms. Ann Myers, HFD-107, Food and Drug Administration, 5600 Fishers Lane, Rockville, Maryland 20857, (301) 443-6004.

Multidisciplinary Management

Head and Neck Cancer

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The multidisciplinary approach to the management of head and neck cancer provides the patient with the most favorable prognosis medically and optimum results cosmetically. Brief case reports illustrate the cooperative evaluations and techniques.

NEW CASES of cancer at all sites in the United States totaled 675,000 in 1976; 24,000 of these were of the pharynx and buccal cavity — 17,000 in males and 7,000 in females.[1] Head and neck skin cancers add significantly to the total, and such prevalence indicates the importance of proper management. Thorough preoperative evaluation, maximally effective therapy, and rapid rehabilitation are the hallmarks of a multidisciplinary approach. These are most effective in the patient with a small lesion. However, the majority of patients with head and neck cancer are heavy drinkers and heavy smokers and, unfortunately, these factors also relate to a poor prognosis.[2] Most early head and neck cancers are detected by dentists; hence the establishment of a multidisciplinary head and neck clinic at Truman Medical Center was ideal because of the proximity of the dental school to in-patient services. Dental students attend the clinic on a regular basis, and staff is drawn from the Department of Oral Surgery and the Oncology Section of the Surgery Department. A patient's preoperative evaluation includes diagnosis by biopsy, determination of extent of disease, multiple biopsies and x-rays if needed, and formulation of optimum treatment by the team. Thus, on admission, appropriate cancer surgery can accommodate and be accompanied by immediate institution of rehabilitative procedures. The following brief cases illustrate the value of this approach.

Recurrent Head and Neck Cancer

Of those head and neck cancers that recur, 80 per cent do so locally. One axiom of head and neck cancer is that recurrences detected early can be treated surgically and control of the cancer re-established. Early recurrence frequently indicates a small tumor that can be re-excised without deforming surgery. Hence, head and neck cancer patients should be seen every two months for two years and then every three months during the third year.

CASE ONE

A 31-year-old female with cancer of the tongue was treated by radon needles and an upper neck dissection. When she was first seen at the Head and Neck Clinic of Truman Medical Center, the tumor had recurred in the submaxillary incision. Excision of the tumor invading skin required excision of the skin of the right side of the neck along with the radical neck dissection, hemimandible, and hemiglossectomy. The defect was successfully closed with an undelayed Mutter flap as illustrated in the following case.



Figure 1. Undelayed Mutter flap used to close defect of upper neck and lower face.



Figure 2. Note overhang of upper lip due to excision of portion of lower lip.



Figure 3. Correction of overhang of upper lip by unabridged Estlander flap.

CASE TWO

A 63-year-old white female was inadequately treated by local excision only for verrucous carcinoma of the gum. After the third recurrence in four months, she was sent for Co^{60} treatment. This stimulated the cancer to grow rapidly through the skin of the jaw. The tumor was removed with a defect — as in the previous case, plus half of the lower lip. *Figure 1* demonstrates the closure of the defect by use of an undelayed Mutter flap.[3] In addition, the problem of disproportionate lip size can be seen in *Figure 2*, and its correction by an unabridged Estlander flap in *Figure 3*. Four months postoperatively, the patient's appearance is acceptable but not as cosmetically desirable as could be expected had she been correctly treated the first time.

These two cases illustrate that recurrent head and neck cancer can be controlled by surgery, but the price is invariably considerable in terms of functional or cosmetic deformity.

Primary Head and Neck Cancer

Cancer of Upper Jaw

CASE THREE

A 53-year-old female was seen in the dental clinic with an extensive verrucous carcinoma of the palate that had invaded into the maxillary antrum. In the multidisciplinary clinic, the oral surgeons obtained a positive biopsy and x-rays of the maxilla, and the team then planned the combined intraoperative treatment-rehabilitation. A maxillectomy was performed utilizing a Weber-Ferguson incision; and reconstruction in the form of a special upper plate with obturator filling the palatal defect was carried out during the same operative period, so that the patient was discharged with no cosmetic or functional deformity.

CASE FOUR

The importance of team effort can be illustrated in the case of carcinoma of the upper jaw in a 67-year-old white male. In the clinic, his staging (multiple biopsies of all suspicious intraoral lesions) and combined approach were planned. A mold was taken of his upper jaw and the model of the planned prosthesis was made based on the plans for resecting the lesion and correcting the expected operative defect. The immediate fitting of the preconstructed prosthesis with its antral obturator occluding the antral opening resulted immediately postoperatively in: (1) normal appearance; (2) normal speech instead of nasal speech; and (3) normal eating habits without regurgitation into the nose.

Cancer of Mandible

CASE FIVE

A 64-year-old female presented with cancer of the left lower alveolar ridge. In the clinic, she was staged and her operative procedure was planned, a Com-



Figure 4. Patient following Commando procedure and stabilization of proximal mandibular fragment with left mandible.

mando procedure with placement of mandibular pins for initiation of immediate rehabilitation. She was admitted to Truman Medical Center and — after direct laryngoscopy and examination under anesthesia — the oral surgeons placed pins as previously outlined, a Commando procedure was carried out, and an acrylic bar put in position. As shown in *Figure 4,* this stabilized the proximal mandibular fragment on the right and prevented deviation of the left hemimandible. The device is worn for as long as three months, and then mandibular reconstruction is carried out.

The importance of the team approach as illustrated by these latter two cases is: (1) aggressive surgery on even small lesions can be effected; (2) no cosmetic deformity; and (3) normal function.

Cancer of Skin

In a similar manner, melanoma of head and neck can be attacked aggressively with excellent cosmetic results.

CASE SIX

A 52-year-old female presented a level V lesion in front of the right ear *(Figure 5).* A large segment of skin attached to the underlying parotid gland was removed in combination with a radical neck dissection. *Figure 6* illustrates the very acceptable cosmetic result one year later.

CASE SEVEN

A 49-year-old female presented with a level IV melanoma on the mid-neck, left side. A neck dissection was performed with an overlying attached piece of skin 10 cm in diameter. Closure was effected with



Figure 5. Level V malignant melanoma in front of right ear.



Figure 6. One year following excision of melanoma in preauricular area with total parotidectomy and radical neck dissection.



Figure 7. Appearance of Mutter flap four months after excision of skin of right lateral neck and neck dissection.

an undelayed Mutter flap. Results four months postoperatively are shown in *Figure 7*.

Conclusion

Aggressive approach to head and neck cancer is being accomplished at Truman Medical Center through the combined efforts of the Department of Oral Surgery and the Oncology Section of the Department of Surgery. The multidisciplinary approach offers the patient both optimal chance of survival and optimal quality of survival.

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YOUR PAYROLL RECORDS AND WORKMEN'S COMPENSATION PAYROLL AUDITS

As you know, the net premium paid for Workmen's Compensation insurance is determined at the end of the policy year by an audit of your actual payroll. The premium you pay at the beginning of the policy year is an estimated premium which is based on an estimated payroll. A plus or minus adjustment is made in premium as a result of the payroll audit at the end of the policy year.

From payroll audits of some association members, it has been found that they are paying higher premiums than necessary for Workmen's Compensation insurance because of the manner in which they compute and record their payroll.

> The premium for workmen's compensation is based on straight-time pay for all hours worked and should not include ½ extra pay for overtime hours worked.

Many members are still using the expensive way (from the Workmen's Compensation premium point of view) to compute and record their payroll. The proper breakdown of payroll may help you reduce premium costs. Example:

WARNING: Do not expect the Workmen's Compensation auditor to refigure your payroll for you, using the correct straight-time method. The Compensation Manual, Page R5, provides, in substance, that the entire remuneration shall be used as a basis for premium. The exception is that if the employer's books and records are kept in such a manner (by employee and class of work) that the amount of overtime pay is readily available in total and in summary for policy period, then the overtime pay (this is the amount earned in excess of the regular pay rate) may be excluded from the premium computation.

The Compensation rules further state that these totals must be available and presented to auditor at time of call. If not, the policyholder is not permitted any later or retroactive adjustment after audit is made.

SPECIAL HOUSE OF DELEGATES MEETING

Sunday, November 12, 1978—10 A.M.—Wichita Hilton Inn

All Delegates Are Urged to Attend!

All Members Are Welcome!

The Philadelphia Chromosome

Its Role in Chronic Granulocytic Leukemia

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The Philadelphia Chromosome, produced by unknown causes through translocation, is a consistent characteristic in a high percentage of chronic granulocytic cases. Its high factor of correlation emphasizes its diagnostic efficacy for this type of leukemia.

THE LEUKEMIAS represent a group of blood disorders characterized by an uncontrolled proliferation of leukocytes and their precursors. Accumulations of these cells are found in bone marrow and peripheral blood samples, and are generally considered to have the same characteristics as malignant neoplasms. They are classified by the dominant cell type and by duration from onset to death with expiration occurring from within a few months in acute cases, to several years in the chronic progressions. The four most common forms are Acute Lymphocytic Leukemia (ALL), Acute Granulocytic Leukemia (AGL), Chronic Lymphocytic Leukemia (CLL), and Chronic Granulocytic Leukemia (CGL). As the names imply, these are characterized by increased numbers of cells from the lymphocytic and granulocytic series in tissues and circulating blood. General symptoms include those of acute infection with severe anemia, hemorrhages and slight or marked enlargement of spleen, lymph nodes, or liver.

One of the most significant advances in the research of these neoplastic disorders occurred in 1960 with the discovery by Nowell and Hungerford of a small, defective chromosome.[1] Named after its location of discovery, the Philadelphia chromosome (Ph^1) has served as a major stimulus for further cytogenetic investigations of human leukemia. The aberration has been found to be a consistent chromosomal abnormality of chronic granulocytic (myelogenous) leukemia, a condition found in no other malignancy.[2]

Nowell and Hungerford developed a technique for the culturing of peripheral blood. Preliminary observations indicated the presence of a minute chromosome in the metaphases of two male patients with CGL. These were thought to be small Y chromosomes. However, studies by Baikie *et al.*[3] revealed the presence of a similar small chromosome in the peripheral blood of two female patients with CGL, thus confirming that the abnormality involved an autosome, rather than the Y chromosome. Subsequent work by the latter group has shown the Ph^1 chromosome to be absent in epithelial cells of otherwise Ph^1 positive patients. Trujillo[4] states that the chromosomal abnormality has proven to be a consistent characteristic in 90-95 per cent of CGL cases, making it a most reliable diagnostic tool. It should be noted that bone marrow is the material of choice. Peripheral blood is not desirable for such determinations, showing a reduced frequency of the Ph^1 chromosome. Thus, a sharp line of distinction is drawn between the typical cases of CGL, invariably Ph^1 positive, and the other myeloproliferative disorders, generally Ph^1 negative.

The specific factors effecting development of the Ph^1 chromosome are unknown at the present time, although several theories do exist. Perhaps the most prevalent theory involves previous patient exposure to irradiation. Durant[2] has noted the increased incidence of CGL four years following the 1945 atomic explosions in Japan, as well as cases of patients treated with high doses of x-ray for ankylosing spondylitis. He is cautious with his observations, however, stating "radiation does cause chromosomal damage, but a link between it and the Ph^1 chromosome has never been demonstrated." Baikie[3] noted similar cases of radiotherapeutic exposure, followed by a diagnosis of CGL. In each instance, however,

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the Ph^1 chromosome is not detected until after a diagnosis of CGL has been made. Thus the question arises: Is the condition of CGL brought about by the Ph^1 chromosome, or does CGL itself cause the chromosomal abnormality?

The debate relating to whether the Ph^1 chromosome has a primary or secondary relationship to the neoplastic state has stimulated much investigation. According to Sandberg,[5] the chromosomal abnormality is not related to radiation therapy as the frequency with which it occurs is not affected by treatment. He sites two cases of patients with leukemia who had received radiation therapy in the past, but in whom no Ph^1 chromosome was detectable. Weiner and Hirschorn[6] have expanded the scope of the problem; they report a limited number of cases in which the Ph^1 chromosome has been found in two generations or among sets of twins, suggesting a possible genetic involvement. Nowell[7] disagrees, however, claiming that the Ph^1 chromosome is "not an inborn change, but rather an induced abnormality." The Philadelphia chromosome is observed before the development of the acute blast phase, indicating that the abnormality is not solely a result of progressive changes in the karyotype occurring in blast crisis.[8] In 1965, Joneja and Stich[9] reported work done with mice inoculated with a Gross virus. They found that abnormal karyotypes were not essential to the development of the leukemia. Conversely, Canellos[10] has reported one case of a 43-year-old white male whose marrow cells were Ph^1 positive for almost six years in the absence of any hematological disease. The patient eventually developed and died from blastic leukemia. The question arises as to the possibility of a "preleukemia" being hematologically undetected in the patient until the initiation of a more noticable chronic phase. Gunz[11] has explored the possibility of a genetic predisposition to leukemia. He notes that in mice, leukemia arises as the result of the combined action of several factors. A single dominant or recessive gene or gene group (Mendelian Pattern) is not responsible for the predisposition, but rather the interaction of other leukemogenic factors (physical agents, viruses) with a fairly large number of genes. This polygenic constitution sets up a threshold that must be crossed before leukemia occurs. Mice strains with low thresholds are more likely to develop leukemia due to this genetic predisposition. High thresholds prohibit the development of leukemia without additional activity of other intrinsic or extrinsic leukemogens. Gunz believes that the genetic background of leukemia in man is similar to that in the mouse, thus advocating a polygenic rather than Mendelian predisposition to the disease. In general, those investigators supporting the secondary relationship of the Ph^1 chromosome to CGL (that is, the neoplastic disorder as the cause of the chromosomal abnormality) refer only to those aberrations that can be detected, and do not take into account the potential existence of undiscernable chromosomal abnormalities. It is felt that if Ph^1 positive and Ph^1 negative chronic granulocytic leukemia are considered as one disease, the Philadelphia chromosome may be thought of as a secondary factor. In this line of thinking, the Ph^1 chromosome either is formed or not formed during the normal progression of the disease. On the other hand, if Ph^1 positive and Ph^1 negative CGL are to be classed as two distinctly different diseases — as is a common practice — the Ph^1 chromosome appears to be a primary cause. The study by Canellos[10] leads to this belief. However, with only one case of this type recorded, the controversy persists as to which condition is the primary cause. The important fact to keep in mind, however, is the high rate of consistency between CGL and the Ph^1 chromosome.

Human chromosomes are identified on the basis of unique banding patterns using quinacrine fluorescence and various Giemsa stains.[8] Individual chromosomes are numbered. The short arm and long arm are designated by "p" and "q" respectively, with (+) and (−) signs indicating whether material has been gained or lost in the abnormality. The Philadelphia chromosome is characterized by a deletion of the long arm of chromosome 22 (22q−). An addition to the long arm of chromosome 9 (9q+) is also seen, the amount being approximately equal to that missing from the Ph^1 (22q−) chromosome. This observation suggests a translocation from chromosome 22 to 9.[8, 10, 12] If the translocation theory is assumed to be true — which would constitute the only specific chromosomal translocation in humans thus far identified — no loss of genetic material would occur. Nowell[7] and Golde[12] suggested that the translocation is produced in a marrow stem cell by radiation or some other mutagen, subsequently involving all three daughter lines.

With a few exceptions, once the Ph^1 chromosome is present in the stem cell line, it persists throughout the course of disease.[13] This has been found to be true even during extremely long periods of remission[2] and during treatment when chromosomal analysis continues to reveal Ph^1 positive cells in the bone marrow. This is not to say that the Ph^1 positive cells totally dominate the cellular population, for normal Ph^1 negative hematopoietic cells have also been found in patients with CGL. Rather, the

myeloid progeny of that Ph[1] positive stem cell acquires some selective growth advantage over the adjacent normal cells. It is theorized that the patient possesses two distinct stem cell lines, one normal and the other bearing the Ph[1] chromosome.[10] During remission, it is assumed that the Ph[1] positive stem cell line exists without excessive proliferation. With the onset of overt leukemia, however, the Ph[1] positive stem cell predominates with the proliferative advantage residing in the granulocytic line. A few cases of conversion of marrow metaphases to predominantly Ph[1] negative cells have been described. The conversions were only found to be transient, however.

The Ph[1] chromosome is present in both bone marrow and peripheral blood of CGL patients. Patient analyses show, however, that the chromosome is found in more than 75 per cent of all aspirated marrow cells processed without culturing, as compared to less than 35 per cent of cultured blood cells.[5] This count may be due to the larger number of younger cells (myeloblasts, promyelocytes, and myelocytes) in the bone marrow. It is these cells that contribute the metaphases used for chromosomal karyotyping.

Chronic granulocytic leukemia is unique among the leukemias in that it progresses in two distinct clinical stages. The first, or chronic phase, is characterized by a greatly increased total granulocyte mass composed largely of mature elements. Many investigators regard this phase to be more of a preleukemic stage. The leukemic cell line is chiefly Ph[1] positive but has an otherwise normal karyotype.[14] It composes but a small percentage of the marrow cells, the remainder being normal. Following the chronic or "preleukemic" stage, the disease progresses into the blastic phase. Here the leukemic cell line possesses a karyotype that is not only Ph[1] positive, but often is accompanied by additional cytogenetic abnormalities other than the Ph[1] chromosome. Immature cells appear and predominate in the marrow (20-30%).[13] Thrombocytopenia and anemia develop, unresponsive to therapy during the chronic phase. In addition, progressive myelofibrosis, splenomegaly, extramedullary tumors, bone pain, fever, chills, an elevated leukocyte alkaline phosphatase, an initial increase in basophils, and cytogenetic evidence of aneuploidy are often observed. This stage appears similar to acute myelocytic leukemia in clinical and morphological symptoms. The CGL patient, however, suffers from an increased resistance to all standard therapeutic agents, the cause of which is unknown. The development of this more complicated karyotype picture is a poor prognostic sign, whether the leukemia is Ph[1] positive or Ph[1] negative. In either case, the patient's survival time is equally limited once he has reached this blastic phase.

Chromosomal studies have been performed on CGL patients during the course of the disease.[14] A fairly systematic chromosomal transition seems to occur in the majority of cases. Most commonly, transitions occur from a marrow containing a leukemic cell line exclusively Ph[1] positive cells, without any other chromosomal changes, to one with some additional karyotypic abnormalities, and then to a marrow consisting exclusively of abnormal Ph[1] positive cells. A reversion in this transition pattern is seldom observed. A reverse transition in bone marrow from one of exclusively abnormal Ph[1] positive cells to one containing some Ph[1] positive cells without other abnormalities is a rare occurrence. If this transition pattern is kept in mind, it is possible that the chromosomal findings could be utilized to determine the stage of the disease. For example, the survival time of patients whose first bone marrow karyotype exhibits only metaphases with a Ph[1] chromosome and other abnormalities averages 7.5 mos, which is significantly shorter than that of patients whose marrow exhibits only metaphases with a Ph[1] chromosome and an otherwise normal karyotype (31.5 mos average). Patients whose marrow contains both categories of cells have an average survival time of 25.5 mos.

In addition to chromosomal constitution, the cell differential of the marrow can also serve as an indication of the patient's prognosis.[15] If karyotypically abnormal Ph[1] positive cells are in the marrow, the survival time is much greater when less than 30 per cent of the cells are myeloblasts and promyelocytes; survival time is much less when more than 30 per cent of the examined cells are of these types. In a like manner, the presence of some normal metaphases in the marrow indicates an early stage of the disease, as they become less frequent with later stages and will disappear entirely if the CGL is not successfully treated. Thus, for a more complete appraisal of the prognosis of the Ph[1] positive CGL patient, an evaluation of both the chromosomal findings and the marrow cell differential is advisable.

Clinical and hematological remissions for limited periods of time have been known to be induced by chemotherapy or splenic irradiation. In cases of a good response, most abnormal parameters, i.e., palpable spleen, high WBC, low hemoglobin, increased myeloid erythroid count, return to normal.[16] The patient is considered to be in complete remission when the bone marrow contains less than 5 per cent

abnormal cells, with a platelet count of more than 100,000/cu mm, and a hemoglobin more than 12 gm/100 ml in the peripheral blood. A partial remission is identified by a decrease to 30 per cent of blast cell population in the bone marrow, with normal peripheral blood readings as above. During a remission, simple microscopic examination of the bone marrow fails to demonstrate any residual evidence of disease. Serial karyotyping studies reveal a decrease in the number of Ph^1 positive cells in the peripheral blood. This coincides with the gradual disappearance of immature myelocytic cells from the circulation, and thus marks the initial effect of the treatment. During a complete remission, no immature hematopoietic elements are found in the peripheral blood, and all Ph^1 positive cells are absent in blood cultures. However, the Ph^1 chromosome persists in the bone marrow at the initial level of incidence, even during the most complete and perfect remissions.[5]

With the Ph^1 positive and Ph^1 negative cells coexisting during remission, why do the Ph^1 positive cells not dominate over the Ph^1 negative cells as they do during the other phases of chronic granulocytic leukemia? Golde[12] concluded that during the remission, the Ph^1 positive clone loses its characteristic proliferative advantage. He demonstrated this phenomenon in vitro as well as in vivo. Liquid cultures from patients with CGL in remission do not show the usual pattern of cell growth; rather cytogenetically normal cells proliferate. However, marrow examinations still demonstrate the presence of Ph^1 positive cells. Golde surmises that as the leukemic clone does not predominate in vitro, it is unlikely that host "suppressive" factors are responsible for the lack of excessive myeloid proliferation in vivo.

Isolated examples of remissions following iatrogenic marrow hypoplasia have been recorded.[17] There is a consensus that this condition favors the reappearance of normal cells in the marrow and, perhaps, leads to a longer remission. This technique is seldom used, however, as the risks of irreversible marrow failure are too great at the present time to allow sufficient testing. The possibility of such treatment being used successfully in the future should not be dismissed. Golde[18] reported one case of a ten-year remission in a CGL patient following a busulfan-induced marrow hypoplasia. (It should be noted that almost all iatrogenic marrow insufficiencies are busulfan-induced.) He hypothesized that a mutation or other change in the Ph^1 positive clone caused these cells to lose their characteristically high proliferative capacity. Unfortunately, the effect is not lasting, as the dominating capacity of the Ph^1 positive cells takes over again when the effects of the marrow hypoplasia diminish.[17]

After a patient has progressed into blastic crisis, the probability of remission is 10 per cent or less. This is true whether his disease is Ph^1 positive or Ph^1 negative. With both conditions presenting an equally poor prognosis, it is important to: (1) seek ways to prevent blastic transformation; and (2) improve methods for inducing a complete remission after the onset of the blastic crisis. It is only in this way that the proliferation of the leukemic cell line, and thus the proliferation of the disease, can be deterred.

Several different methods of blastic therapy are presently in use as treatment for CGL. Chemotherapy is most frequently utilized.[13] Prednisone and vincristine are often combined. Drug protocol calls for vincristine sulfate, 2.0 mg/sq m body surface area, administered intravenously on a weekly schedule. Prednisone, 60 mg/sq m orally/day is added and maintained for two weeks, at which time a gradual tapering of the dose is begun. The therapy is continued as long as progressive hematological improvement is noted. This treatment has been known to produce remissions in as many as 30 per cent of CGL patients in isolated studies. However, this is an unusually high response rate, whereas more often the response is in the range of 10-12 per cent. For this reason, the use of this type of chemotherapy for remission induction, followed by adequate maintenance therapy, can significantly prolong remission. It is also used to achieve a second remission in patients who have relapsed. Other drug combinations have been found to be effective in producing a good response. Perhaps the best is COAP (cyclophosphamide, vincristine, *arabinosyl* cytosine, and prednisone). Other drug combinations, such as DOAP (*daunomycin,* vincristine, *arabinosyl* cytosine, and prednisone), COPP (*methylglyoxal* bis guanyl hydrazone, vincristine, prednisone, and 6-mercaptopurine), or the use of single agents have been found to be less effective.

Treatment involving the spleen has occasionally been executed with some degree of success. The Medical Research Council reports that splenic irradiation has been found to produce a median survival of up to 120 weeks (counted from the first day of treatment).[18] A more radical procedure, splenectomy, can also initiate remission,[16] especially after the patient has entered blastic crisis. This surgical procedure is difficult due to numerous bleeding problems encountered in the CGL patient. To date, the effectiveness of this treatment has not been completely evaluated.

The present approaches to the treatment of pa-

tients with CGL are only palliative. The Ph[1] chromosome persists, even throughout therapy and remissions.[4] This can be equated to survival of the cells containing the cytogenetic mark of CGL. The only definitive cure is permanent eradication of the Ph[1] positive clone of cells from the bone marrow. Unfortunately, this objective has not been achieved, according to the currently-reported advancements. A possible approach is toward more aggressive therapy earlier in the course of the disease. However, the risk of fatal aplasia in the patient discourages such a move. Current study in the area of cell kinetics and chemotherapy of myeloblastic leukemias may well provide the answer to this problem in the near future.

The absolute specificity of the Philadelphia chromosome for CGL is questioned by the existence of a scattered number of cases with the classic features of CGL but without the Ph[1] chromosome. The incidence of this has been reported to be as high as 12 per cent,[2] but usually is much less. The exact nature and source of the Ph[1] negative cells in the bone marrow remain unknown. It is speculated that they arise from stem cells that, although present in the bone marrow in CGL, only proliferate in very unusual circumstances. Alternatively, they may be of extra-myeloid origin, arising from lymphocytes, colonizing the bone marrow with Ph[1] negative cells under a stimulus provided by marrow insufficiency.[18] Whatever the origin of these cells, they persist in the Ph[1] negative CGL patient in relatively unchanged proportions throughout the course of the disease.

Although the classical symptoms of CGL are present in the Ph[1] negative patient, several atypical features also exist. According to the National Cancer Institute, CGL patients with Ph[1] negative disease have a higher median age (60 vs 42 yrs) and are diagnosed at lower median leukocyte (41,500 vs 114,000) and platelet counts (180,000 vs 388,000). They exhibit a more pronounced immaturity in the bone marrow and peripheral blood cells. The response to chemotherapy is generally poor, especially relating to busulfan and 6-mercaptopurine therapy. The clinical course of the Ph[1] negative patient is one of extremely rapid deterioration, complicated by progressive anemia and thrombocytopenia. Death usually occurs within one year, as compared to a median survival time of 45 mos for the Ph[1] positive CGL patient. However, all patients die with hematological features of blastic crisis, indicating a fairly normal chromosomal transition for the disease. In general, the absence of the Ph[1] chromosome in patients with the hematological characteristics of CGL is a poor prognostic sign. Because of these differences, Ph[1] negative CGL is sometimes considered to be a distinct clinical entity from Ph[1] positive CGL. Thus the presence or absence of the chromosome in CGL has obvious therapeutic and prognostic implications.

The basis for the varying symptoms between Ph[1] positive and Ph[1] negative CGL is unknown. One interesting observation has been made, however, in relation to the chromosomal patterns. If the Ph[1] chromosome is assumed to be a translocation from number 22 to number 9, then leukemic cells with or without the Ph[1] chromosome have approximately the same amount of chromosomal material. Thus, differences in the clinical course cannot be caused by an appreciable loss of chromosomal material.

In general, the Philadelphia chromosome can be used as a fairly consistent indicator of CGL. A few scattered cases of unusual forms of other Ph[1] positive myeloproliferative disorders have been reported, but these are usually hematopoietic conditions allied to CGL, *i.e.*, AML, polycythemia vera, myeloid metaplasia.[19] Perhaps these cases indicate that the mere presence of the Ph[1] chromosome does not positively identify a case of typical CGL, yet the bulk of evidence recorded does not support this theory. Far more plausible is the possibility that, in such cases, either the leukemia is actually masquerading throughout its course as another myeloproliferative disorder, or the patient has an additional disease.[20] Whatever the answer may be, cases of this type are so few in number that they should cause relatively little concern. Their presence should be noted, however.

In the past, the diagnosis of chronic granulocytic leukemia has been based on clinical and morphological symptoms. The karyotyping procedure is not done routinely, perhaps because of the expense involved and relative newness of the technique. The Philadelphia chromosome has been demonstrated to have a high factor of correlation with CGL. Although it is not known if the translocated chromosome is a primary or secondary causative agent to the disease, it has been found to appear in 90-95 per cent of all CGL cases. Occasionally, a diagnosis between CGL and other closely related myeloproliferative diseases is nearly impossible without the diagnostic aid of a karyotyping for the presence of the Ph[1] chromosome. The karyotype also aids the physician in determining the approximate stage of the disease and thus an appropriate course of treatment, as well as a relative survival time for the patient. With these recent developments at hand, it appears likely that the presence of the Philadelphia chromosomes will continue to play an increasingly

important role in the diagnosis and treatment of chronic granulocytic leukemia.

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Case Reports

MULTIPLE PRIMARY BRONCHOGENIC CARCINOMA

Multiple primary bronchogenic carcinomas are rare. Rohwedder and Weatherbee,[1] in a review of 926 patients with lung cancer, found five cases — an incidence of 0.5 per cent. Our experience of six cases — including an instance of previously unreported association of giant cell and undifferentiated small cell bronchogenic carcinomas — prompts this review.

Table 1 lists the clinical and anatomical features of the six cases. All were men, a reflection of a predominantly male hospital population. The age range was 50-80 yrs with a mean of 65 yrs.

Duration of life from onset of symptoms ranged from 6-12 mos, with a mean of 8.5 mos. All six patients died of widespread metastases. This rapid evolution is characteristic of undifferentiated carcinoma which was present in four of the patients.

In *Patient 1,* a squamous cell bronchogenic carcinoma was found by bronchial biopsy, and was treated with right upper lobectomy followed by Cobalt-60 therapy (3,500 rads). At autopsy, only remnants of squamous cell carcinoma were found in the remaining right lung. Microscopic examination revealed the largest single tumor — which was localized to the middle lobe bronchus — to be an undifferentiated small cell carcinoma. All the metastases present were from the undifferentiated tumor; none were from the original tumor diagnosed six months before death as squamous cell bronchogenic carcinoma.

The first tumor of *Patient 2* was found to be too extensive for radical therapy, and only a diagnostic biopsy was obtained revealing squamous cell bronchogenic carcinoma. Due to the patient's terminal condition, only symptomatic and supportive treatment were given. Autopsy disclosed a secondary primary tumor diagnosed as undifferentiated small cell carcinoma. Diffuse metastases of the squamous cell carcinoma were seen and only hilar metastasis of the undifferentiated small cell carcinoma was present.

The case of *Patient 3* was very interesting as it is apparently the first reported giant cell bronchogenic carcinoma to be associated with an undifferentiated small cell carcinoma *(Figures 1, 2).* Diffuse metastases were seen of the undifferentiated small cell carcinoma, and hilar metastases were detected for the giant cell bronchogenic carcinoma.

In *Patient 4,* biopsy of the left upper lobe bronchus revealed undifferentiated squamous cell carcinoma. Because of the terminal condition of the patient, only symptomatic and supportive treatment were given. Autopsy disclosed a peripheral adenocarcinoma of the right lung, and diffuse metastases were found of both types of tumor.

At the time of admission, *Patient 5* was already cachectic and terminal, and no treatment was given. Autopsy disclosed a squamous cell carcinoma of the left upper lobe and a large cell undifferentiated carcinoma of the left lower lobe. Diffuse metastases of both tumors were present.

In *Patient 6,* roentgenogram of the chest revealed a possible tumor of the right lung, but the patient refused further diagnostic procedure or treatment. Three months later he developed a pathological fracture of the left humerus. A biopsy of the area of fracture revealed metastatic undifferentiated carcinoma, probably from the lungs. Because of his poor physical condition, only symptomatic and supportive treatment were given. Autopsy disclosed an undifferentiated large cell bronchogenic carcinoma of the middle lobe of the right lung and an oat cell carcinoma of the lower lobe of the same lung. Widespread metastases of the large cell bronchogenic carcinoma were seen, but there were no metastases of the oat cell carcinoma.

Table 1

ANATOMIC AND CLINICAL DATA ON SIX PATIENTS WITH MULTIPLE PRIMARY BRONCHOGENIC CARCINOMAS

Patient	*age*	*Cigarette Smoking*	*Onset of Symptoms to Time of Death*	*First Primary*	*Site*	*Diagnosis Made By*	*Treatment*	*Metastases*	*Second Primary*	*Site*	*Diagnosis Made By*	*Metastases*
1	59	40 cig. a day	6 months	Squamous cell carcinoma	Right upper lobe bronchus	Biopsy	Right lobectomy Cobalt 3,500 Rads	None	Undifferentiated small cell carcinoma	Lower lobe right lung	Autopsy	Diffuse
2	74	20 cig. a day	8 months	Squamous cell carcinoma Giant cell carcinoma	Right middle lobe bronchus	Autopsy	None	Hilar lymph nodes	Undifferentiated small cell carcinoma	Right lung	Autopsy	Diffuse
3	50	35 cig. a day	12 months	Squamous cell carcinoma	Upper lobe left lung	Diagnostic thoracotomy	Symptomatic & supportive	Diffuse	Undifferentiated small cell carcinoma	Right middle lobe	Autopsy	Hilar lymph nodes
4	70	25 cig. a day	10 months	Undifferentiated squamous cell carcinoma	Left upper lobe bronchus	Biopsy	Symptomatic & supportive	Diffuse	Adenocarcinoma	Peripheral right lung	Autopsy	Diffuse
5	58	40 cig. a day	9 months	Squamous cell carcinoma	Left upper lobe	Autopsy	None	Diffuse	Undifferentiated large cell carcinoma	Lower lobe left lung	Autopsy	Diffuse
6	80	35 cig. a day	6 months	Undifferentiated large cell carcinoma	Right middle lobe	Biopsy	Symptomatic & supportive	Diffuse	Undifferentiated small cell carcinoma	Lower lobe right lung	Autopsy	None

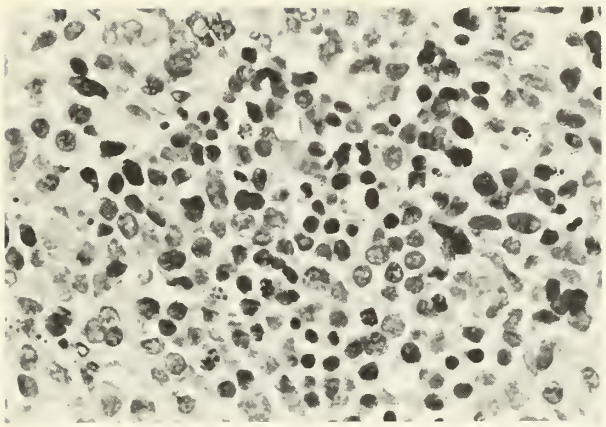


Figure 1. Patient 3 microphotograph of undifferentiated small cell carcinoma. Magnification ×400. H&E stain.

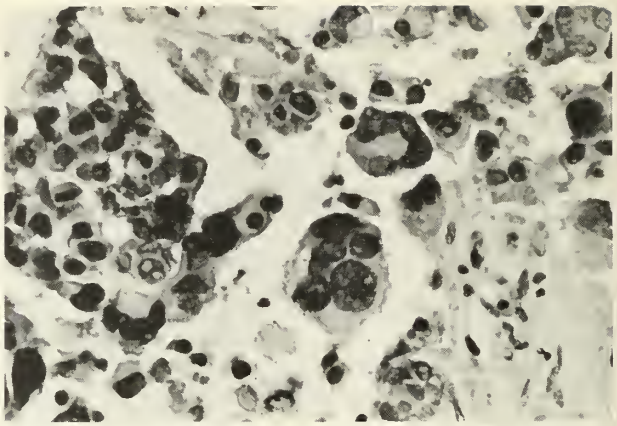


Figure 2. Patient 3 microphotograph of giant cell bronchogenic carcinoma. Magnification ×400. H&E stain.

All six patients were cigarette smokers with an average daily consumption of 32.5 cigarettes.

Discussion

Multiple malignancies arising in one or more organs are fairly common. They are often seen among tumors of breast,[2] ovaries,[3] skin,[4] and digestive tract,[5] but are rarely noted among lung tumors. The reported incidence varies anywhere from 0.5 per cent[1] to 7.8 per cent.[6] Struve Christenson,[6] on reviewing the literature, found 70 patients with bilateral primary bronchogenic carcinoma, of which 46 were of the same histologic type. In view of the difficulty in differentiating primary from metastatic tumors, especially in the lungs, tumors of identical cell types should not be classified as two different primary tumors. Taking this precaution into consideration, the incidence of multiple primary bronchogenic carcinomas should be close to the figure of 0.5 per cent given by Rohwedder *et al.*[1]

Billroth and Winiwarter in 1887[7] introduced criteria for establishing beyond reasonable doubt the presence of a secondary primary tumor: (1) the tumors should be in separate locations; (2) they should differ in histologic appearance; and (3) each tumor should produce its own metastases. Most authors agree that these critieria are overly strict, and that those of Warren and Gates[8] are more practical: (1) each tumor must present a definite picture of malignancy; (2) each tumor must be distinct; and (3) the possibility of one tumor being metastatic from the other must be excluded. Epidermoid bronchocarcinomas can be considered to be of primary origin only when seen to be arising from bronchial mucosa. It is not practically possible to determine the existence of multiple primary undifferentiated small cell, large cell, or adenocarcinomas in the lung because of the nature of tumor growth. Metastases localized in mucosa and submucosa of a bronchus may be impossible to differentiate from second primary tumors of similar types.[9]

Garland *et al.*[10] studied the rates of growth and apparent durations of bronchocarcinomas and found that growth to a diameter of 2.0 cm requires 10.8 years for squamous cell carcinoma, 11.2 years for undifferentiated carcinoma, and 18.6 years for adenocarcinoma. It follows that clinical diagnosis of a single bronchogenic carcinoma does not rule out the possibility of a second smaller tumor not yet clinically apparent. Of the multiple bronchogenic carcinomas reported in the literature, 84.2 per cent have been synchronous and 15.8 per cent asynchronous.[11] The most frequent combination was squamous cell carcinoma with oat cell carcinoma. The tumors were unilateral in 20 per cent and bilateral in 74 per cent (in 6% the location was not specified), with equal distribution between the two lungs.[1] In the six cases presented here, four were unilateral combinations and two were bilateral. All were of different cell type, with predominance of squamous cell carcinoma, and all the tumors were asynchronous.

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References may be obtained from the authors.

NORMOURICEMIC ACUTE POLYARTICULAR GOUT

Hyperuricemia has long been accepted as the rule in the vast majority of patients with gout.[1, 2] Definitive diagnosis, however, depends on the detection of monosodium urate crystals in the synovial fluid. The clinical presentation and serum uric acid levels of the following case might cause difficulty in establishing the diagnosis.

Case Report

An 80-year-old man presented with painful swelling of his left knee, both ankles, and right wrist. He gave no history of gouty attack and had not been taking aspirin or other medications. His temperature was 38.9C. Results of laboratory examination were WBC, 13,000/cu mm; serum urate concentrations, 4.7 and 4.6 mg/dl. The left knee was aspirated and ten milliliters of synovial fluid removed. Using the technique of compensated polarized light microscopy, abundant monosodium urate crystals were seen inside the leukocytes of the aspirated fluid. Colchicine was not initiated due to the patient's complaints of nausea and loose bowels. His arthritic symptoms responded rapidly to indomethacin. Subsequent serum acid concentrations were within normal limits.

Discussion

Acute polyarticular gout has been considered a rare form of the disease, particularly unusual as a first manifestation.[3] Polyarthritis, together with normal serum urate concentrations,

may cause the clinician to overlook the possibility of acute gout. Observations in one series revealed that 32 patients (38%) with definite and 68 patients (23%) of those with probable cases of acute polyarticular gout were normouricemic at the time of attack.[4] In the same series, it was observed that the administration of pharmacologic agents having uricosuric activity preceded polyarticular gout more frequently in normouricemic than in hyperuricemic patients.

The diagnosis of gout should be entertained in asymmetric polyarthritis with or without foot involvement or normouricemia, and should then be confirmed by synovial fluid examination.[5]

Summary

A case of acute polyarticular gout with normal serum uric acid levels has been presented. Review of the literature reveals that hyperuricemia is not, as has long been stated, the rule in the diagnosis of acute gouty attack. Normouricemia should not mislead the clinician from the diagnosis of gout, particularly when the patient presents with polyarthritis. Synovial fluid analysis should be done to confirm the diagnosis.

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References may be obtained from the authors.

SYSTEMIC LUPUS ERYTHEMATOSUS ASSOCIATED WITH ADRENAL INSUFFICIENCY

Idiopathic Addison's disease (IAD) is known to be associated with several autoimmune disorders such as pernicious anemia, Hashimoto's thyroiditis, hypoparathyroidism, and diabetes mellitus.[1] However, the association of systemic lupus erythematosus (SLE) with IAD is extremely rare. In 1973, Eichner *et al.*[2] described the association of Addison's disease and SLE in a 26-year-old female in whom adrenal insufficiency developed four years prior to confirmation of the diagnosis of SLE. There are a few earlier reports in the literature alluding to such an association, but the diagnosis of either IAD or SLE was not definitely established in those reports.[3-6]

Case Report

A 62-year-old Caucasian male was admitted to the Topeka Veterans Administration Hospital in January 1977 with the complaints of progressive generalized weakness and vague abdominal pain associated with recurrent nausea and vomiting of three months' duration. The patient's past history included recurrent bilateral pleuritic chest pain and arthralgias involving multiple joints. He had also lost 20 kg during the six months prior to this admission. There was no known exposure to drugs or chemicals.

A few weeks earlier, he had been hospitalized elsewhere with the same presenting complaints. During that admission he was found to have persistent hyponatremia; serum sodium levels ranged between 119-126 mEq/liter and potassium levels fluctuated between 3.9 to 5.6 mEq/liter. Examination on admission revealed the following: hematocrit, 35.5%; hemoglobin, 11.4 gm/100 ml; WBC, 8,000/cu mm, with 78% neutrophils, 16% lymphocytes, 2% eosinophils, and 4% monocytes. Platelet count was reported to be adequate. Chest x-ray revealed pleural thickening at the bases, but was otherwise unremarkable. Two weeks following admission, examination revealed: hematocrit, 26.5%; WBC, 4,450/cu mm, with 62% neutrophils, 33% lymphocytes, 3% eosinophils, and 2% monocytes. Platelet count was decreased. Progressive pancytopenia soon became evident; a few days later hematocrit further dropped to 23%; WBC to 2,900/cu mm, with 49% neutrophils, 40% lymphocytes, 8% eosinophils, and 3% monocytes. Platelet count was 40,000/cu mm. The patient was transfused with several units of packed cells during this admission.

On admission in January 1977 to the Topeka Veterans Administration Hospital, the patient was found to be emaciated, weighing only 45 kg. Skin hyperpigmentation was more pronounced over exposed areas such as the face and dorsal aspect of both arms. Skin turgor was poor and he was clinically dehydrated, with pulse, 90; and blood pressure, 110/70 mm Hg. The thyroid gland was not enlarged; liver, spleen, and lymph glands were not palpable. Examination of the chest was unremarkable. Initial laboratory data showed hematocrit, 30%; WBC, 2,888/cu mm, with 57% neutrophils, 37% lymphocytes, and 6% eosinophils; platelet count, 20,000/cu mm; reticulocyte count, 10%; ESR, 62 mm/hr; serum sodium, 122 mEq/liter; serum potassium, 5.6 mEq/liter.

Cachexia, increased pigmentation, and persistent hyponatremia with kyperkalemia indicated adrenal insufficiency on admission, and serum ACTH and 24-hr urine specimens were obtained for 17-hydroxy and 17-ketosteroid level determination. However, a few days after admission the patient developed an acute upper respiratory tract infection precipitating an acute adrenal crisis. The Addisonian crisis was managed with prompt intravenous fluid infusion and dexamethasone 4 mg administered parenterally. A simultaneous ACTH stimulation test was performed. Plasma cortisol levels were measured prior to and at 30 and 60 minutes after aqueous ACTH 40 units was administered intravenously. There was no change in the plasma cortisol level from the baseline value of 4.7 micrograms/dl (normal 6-27 micrograms/dl). The patient was started on prednisone 15 mg orally daily when the acute adrenal crisis had been stabilized. Results of preliminary investigations performed earlier for adrenal insufficiency revealed: serum ACTH level, 194.6 picograms/ml (normal 10-80 pi̧cograms/ml); 24-hour urinary 17-OHCS, 0.7 mg (normal 2-10 mg); and 17-KS level, 3.6 mg (normal 9-25 mg). These findings confirmed the diagnosis of primary adrenocorticol insufficiency. Serum thyroxin and triiodothyronine levels were normal. Bone marrow aspirate and biopsy revealed hypercellular bone marrow with increased number of megakaryocytes. Normoblastic erythroid hyperplasia with normal granulocytic development was noted. Focal areas of lymphocytic hyperplasia were observed. No tumor infiltration or granuloma were present. Investigations for tuberculosis and fungal diseases were negative.

The cause of his pancytopenia was pursued further and underlying SLE was confirmed by the following laboratory findings: anti-nuclear antibodies (ANA) positive in a titer of 1 : 1280 with solid pattern; LE preps positive and antibodies to double-stranded DNA found to be in a concentration of 66 units/ml (normal <15 units/ml); serum C_3 level, 60 mg/dl (normal 80-140 mg/dl); C_4 level 12 mg/dl (normal 20-40 mg/dl). Immunofluorescent study of the patient's skin revealed characteristic granular deposition of IgM at the dermo-epidermal junction. Serum protein electrophoresis showed a polyclonal increase in gammaglobulin fraction.

Plasma fibrinogen, fibrinogen split products, partial thromboplastin time, and prothrombin time were normal. There was no evidence of circulating anticoagulants, and there was no spontaneous bleeding during his entire clinical course. Urinalysis showed no proteinuria or casts. Other test results revealed: serum creatinine and BUN, normal; serum total biliru-

TABLE I
RESULTS OF STANDARD ACTH STIMULATION TEST,

Date	Test	Plasma Cortisol (N: 6-27 microgm/dl)	Urinary 17-OHCS (2-10 mg/24h)	Urinary 17-KS (9-25 mg/24h)
11/30/77	Daily ACTH 40 units I.V. infusion over 8 hrs for 3 days	13.5	1.0	10.3
12/1/77		—	1.2	15
12/2/77		—	2.4	10.3
12/3/77		12	2.3	13.3

bin, 2.6/100 mg and direct bilirubin, 0.8/100 mg; SGOT, 134 mu/ml (normal 7-40 mu/ml); LDH 285 mu/ml (normal 100-200 mu/ml); serum haptoglobulins, 30 mg/dl (normal 100-300 mg/dl); serum B_{12} and folate levels, normal. Antithyroid and antiadrenal antibodies were negative.

Hematological improvement was noted a few days after initiation of steroid therapy. Eight weeks later, examination showed: platelet count, 188,000/cu mm; WBC, 8,600/cu mm, with 72% neutrophils, 18% lymphocytes, 3% eosinophils, and 7% monocytes; hematocrit, 38%. On a maintenance dose of 15 mg prednisone/day, the patient's complete blood count has remained normal during the entire follow-up period of more than one year.

Several months after the diagnosis of IAD, re-evaluation with a prolonged ACTH stimulation test of the patient's adrenal reserve yielded results summarized in *Table I*.

Discussion

Well-documented association of Addison's disease with SLE similar to the case reported here is extremely rare in the literature. However, symptoms and signs of adrenal insufficiency — such as weight loss, nausea and vomiting, and hyperpigmentation — often occur in SLE alone without any evidence of concomitant Addison's disease.[7]

The single most striking manifestation of SLE in this patient was severe pancytopenia which responded to a minimal dose of prednisone. Past history of arthralgias and recurrent pleuritic chest pain could be secondary to SLE; however, no clinical evidence of renal involvement or cutaneous manifestations of vasculitis was observed.

It is highly probable that underlying SLE is causally related to IAD in this patient. It is of interest that no antiadrenal antibodies could be demonstrated, though they were found in 60-75 per cent of cases of IAD.[1,8] It is speculated that, as in the case reported by Eichner *et al.*,[2] ischemic involvement of adrenal cortex due to localized vasculitis might have contributed to adrenal insufficiency in this patient as well. It is probable that the slight improvement in the basal plasma cortisol level and 24-hour urinary 17-OHCS and 17-KS levels noted after several months of prednisone therapy reflect partial recovery of adrenal function resulting from amelioration of vasculitis by prolonged steroid therapy. Adrenal insufficiency resulting from vasculitis and endarteritis in other clinical disorders have been reported in the literature.[9,10] Although antiadrenal antibodies were negative, the possibility of autoimmunity, a common denominator for both SLE and IAD, could not be disregarded as an explanation for adrenocortical insufficiency in this patient. In any event, it is very unlikely that this case represents simple coexistence of these two diseases. It is suggested that the possibility of underlying SLE should be kept in mind in patients presenting with IAD.

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References may be obtained from the authors.

ASEPTIC CAVITARY PULMONARY INFARCTION

Aseptic cavitary pulmonary infarction is rare. Only 19 cases could be found in a scan of the literature. The diagnosis of this condition and its differentiation, particularly from tuberculosis and malignancy, may be extremely difficult without biopsy or autopsy. One such case, with diagnosis confirmed by biopsy, is presented here. This further confirms the existence of the condition as a clinical entity with emphasis on certain distinguishing features of the disease.

Case Report

A 54-year-old Caucasian male, a long-time nursing assistant by profession, was admitted to the Topeka Veterans Administration Hospital on July 14, 1977, for evaluation of a lesion found in the right lung. This lesion was detected in a neighboring hospital on routine chest roentgenography prior to surgery for bilateral inguinal hernias, and he was referred to this VA Hospital. The patient denied past history of any significant cardiopulmonary disease. His annual chest x-rays and tuberculin skin tests had always been negative. System review revealed that the patient had undergone surgery for duodenal ulcer in 1959, enucleation of the right eye following an injury approximately 45 years earlier, and removal of a skin cancer from the nose in mid-1975. On further questioning, the patient admitted that he had experienced a flu-like attack about three weeks prior to admittance, but he did not attach much significance to it. He had been smoking one package of cigarettes/day for the past 40 years.

Physical examination showed a thin individual looking older than his age with no particular complaints or distress; clubbing of fingernails, 1+; prosthetic right eye; heart and lungs, unremarkable; liver, just palpable; bilateral inguinal hernias; mobility of the spine, impaired; slight generalized tendon hyperreflexia. Some varicosities in the left lower limb were noted, but there was no obvious evidence of venous thrombosis in the lower limbs.

Laboratory workup including complete blood count, automated blood profile, urine examination including culture, and serology revealed no abnormality. Other findings included: sedimentation rate, 39 mm/hr; repeated sputum examinations and cultures, negative; sputum for cytology, Class I and Class II; skin test for tuberculosis — intermediate and second strength of PPD — negative; skin tests and complement fixation tests for fungi, normal; pulmonary function tests, normal; EKG, unremarkable. X-rays taken four months prior to admission showed no lesion, but calcification in the right hilar area was identified. X-rays of the chest on this admission showed dense infiltrative lesion on right upper zone; Lordic view showed a well-defined cavity at the right apex with infiltrative lesion *(Figure 1)*. Tomograms of the right lung revealed cavitations with relatively thin walls or pneumatoceles on the right apex with an ill defined



Figure 1. X-ray showing well-defined cavity at the right apex with infiltrative lesion.

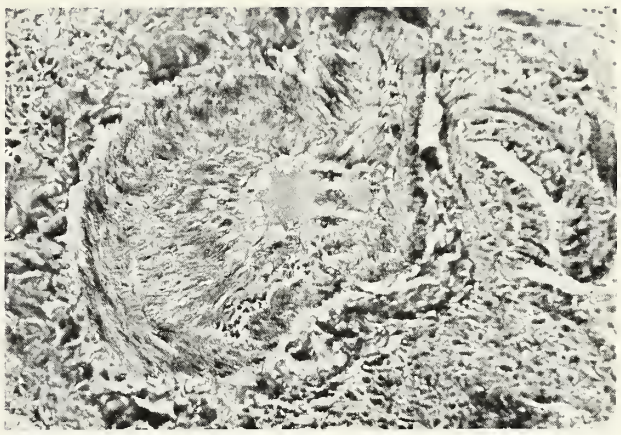


Figure 2. Medium-sized artery obstructed with a clot undergoing organization, liquefaction, and absorption. van Gieson reticulin stain.

soft tissue density at the level of anterior portion of the second rib and a soft tissue line extending from this mass to the hilum. The radiological impression favored infection or neoplasm. Fiberoptic bronchoscopy revealed no abnormality, and bronchial washings were negative for any pathogen. However, cytology of the brush biopsy from the apical segment of the right upper lobe revealed Class V cells.

In view of the Class V malignant cells observed by brush biopsy, the right chest was explored. A firm, 3-cm mass, densely adherent to the chest wall, was found in the apex and was excised. Frozen biopsies of the adherent chest wall, apex of the right lung and several right hilar lymph glands showed no evidence of malignancy, but as malignancy could not be definitely ruled out, the right upper lobe was removed. Resected right hilar lymph glands showed reactive lymphadenitis. Results of examination of the resected lung tissue for pathogens — including acid-fast bacillus and fungi — were negative. Biopsy of the resected lobe revealed the following: (1) organizing infarction with cavitation (*Figure 2* shows a van Gieson reticulin stain manifesting a medium-sized artery obstructed with a clot undergoing organization, liquefaction and absorption); (2) interstitial fibrosis; and (3) atypical cuboidal and squamous metaplasia. The patient made an uneventful recovery after surgery.

Comments

It is generally accepted that true pulmonary infarcts take a few weeks to several months to undergo resolution with remnants of a scar that may at times be fine and difficult to detect. Cavitary changes noted in some cases of bland pulmonary infarction have been mostly attributed to secondary infection with resultant pulmonary abscess formation. In the large series studies recorded in the literature, Chester and Krause[1] noted 17 cases of pulmonary abscess among 333 pulmonary infarcts. Spain and Moses,[2] in a study of more than 100 cases of pulmonary infarction, noted liquification necrosis in only five cases, four of which showed evidence of previous lung infection. In Mayo Clinic observations of more than 550 cases of pulmonary infarcts, Levin *et al.*[3] noted frank abscesses in 4.2 per cent of cases. The secondary infection of bland pulmonary infarcts as observed in the Mayo Clinic report and by others is characterized by foul and purulent sputum, days or weeks after the onset of infarction, fever, tachycardia, leukocytosis, intractability of cardiac failure, elevation of sedimentation rate, and persistance of ill defined symptoms such as malaise. It has also been noted[4] that important diagnostic x-ray signs of a sterile — as opposed to a septic — cavity are constant size, irregular outline, and absence of a clear fluid level. It is important to remember that infarcts may sometimes give a false positive roentgenographic impression of cavitation[5] due to central zone of ischemia surrounded by a zone of hyperemia and edema.

The pathogenesis of aseptic cavitation is not clear. This has been suggested to be due to aseptic liquification necrosis occurring in infarcts, usually larger than 4 cm in diameter.

The case presented here demonstrated roentgenographic and pathological evidence of cavity formation without fluid level within bland pulmonary infarct. There was no evidence of broncho-pulmonary infection during the entire episode. Prior to the lobectomy, there was no clue to suggest aseptic cavitary pulmonary infarction. Detection of Class V malignant cells on brush biopsy turned the diagnosis in favor of malignancy. The true nature of the disease — aseptic cavitary pulmonary infarction — was revealed only after resection of the diseased lobe and histological and cultural studies of the biopsied materials. It is interesting to note that Scoggins *et al.*[6] has recently published a report of three cases in which positive cytological diagnoses of carcinoma of the lung were made by an experienced cytologist. During surgery, each patient was found to have a pulmonary infarct with no evidence of carcinoma. In two of the three patients, left upper lobectomy was performed. These cases are similar in this respect to our patient, although the lung lesions were not cavitary in nature.

Study of the literature reveals a total of 19 cases of aseptic cavitation of pulmonary infarction so far reported; 17 of these cases have been listed by Grieco and Ryan[4] including one case of their own and subsequently two cases by Scharf *et al.*[5]

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References may be obtained from the authors.

T_4 TOXICOSIS IN GRAVES' DISEASE

Thyroxine (T_4) toxicosis, a hyperthyroid state in which serum T_4 level is elevated without elevation of serum triiodothyronine (T_3) concentration, is a very rare entity. Its existence has been questioned,[1] despite a few case reports.[2, 3] It has been suggested that T_4 toxicosis may be a normal biochemical finding in elderly women.[1] Recently, Sobrinho and co-workers[4] reported T_4 tox-

icosis in 5 of 15 patients with iodine induced thyrotoxicosis. All of their 47 patients with Graves' disease had elevated serum T_3 concentrations. Reported here is a case of T_4 hyperthyroidism in Graves' disease.

Case Report

A 78-year-old black man presented with general weakness and difficult swallowing. He had been unable to eat for three weeks prior to admission. He reported a 14 kg weight loss in two months. He gave a history of having had a "liver condition." Physical examination revealed an elderly, cachectic, nervous man. Pulse rate was 120/min with occasional ectopic beats. The skin was smooth, warm, and moist; there was no ophthalmopathy. The thyroid gland was diffusely enlarged, estimated weight, 55 gm. A loud bruit was audible over the thyroid. There was mild pitting edema of both ankles. Laboratory examination revealed the following: serum T_4, 14 mcg/dl (normal 5.0-13.4 mcg/dl); resin uptake test, 57.3% (normal 35-45%); free thyroxine index (FTI), 8.02 (normal 1.75-6.03); serum T_3 concentration, 160 ng/dl (normal 80-220 ng/dl); 24-hr thyroidal ^{131}I uptake, 81.5% (normal 5-35%). Thyroid scan revealed an enlarged thyroid with uniform uptake of ^{131}I. Liver function tests revealed: serum total protein, 8.0 gm/dl; albumin, 2.0 gm/dl; total bilirubin, 3.6 mg/dl; direct bilirubin, 1.8 mg/dl; and alkaline phosphatase, 220 mu/ml (normal 30-120 mu/ml). The patient was treated with prophylthiouracil and digoxin. One week after the treatment was initiated, serum T_3 was 180 ng/dl. The patient was discharged in improved condition one month after admission. Two months later, serum T_4 was 7.9 mcg/dl and FTI was 4.66.

Discussion

Hyperthyroidism is usually associated with elevated serum T_4 and T_3 concentrations. Occasionally (4-30% of cases) only T_3 toxicosis is present. In rare instances,[2-4] T_4 toxicosis does occur. This situation may result from several conditions: hyperthyroidism with coexistent non-thyroidal diseases that have impaired peripheral conversion of T_4 to T_3 such as hepatic and renal disorders; starvation; malignant disease; protein-calorie malnutrition; carbohydrate deprivation; corticosteroid therapy; and surgery.[7] These situations have been shown to result in a decrease in serum T_3, and an increase in reverse T_3 (rT_3).[8-11]

The findings in the patient presented here may have resulted from diminished extrathyroidal conversion of T_4 to T_3 due to his non-thyroidal conditions — liver disease, cachexia, starvation, and senescence.[12] There is a marked increase in T_3 secretion in hyperthyroidism,[13] but under the foregoing conditions the thyroidal secretion of T_3 or thyroidal conversion of T_4 to T_3 prior to secretion — if the capability exists — may be partially inhibited.

Under conditions of abundant iodine supply, the autonomous thyroid tissue frequently present in old goiters secretes T_3 and T_4 in proportions similar to the one observed in euthyroid individuals from non-edemic areas. Therefore, the appearance of T_4 toxicosis is a likely event.[4] But with no history of excessive iodine intake in this patient, his thyroid gland was diffusely enlarged, not multinodular.

The incidence of T_4 toxicosis is not known. A survey by Hadden *et al.*[14] suggested that T_4 toxicosis does exist. Two instances of T_4 toxicosis in 90 hyperthyroid patients (2.2%) were reported in their series. They concluded that these patients represent merely segments of the borderline area. The present case should not be considered a borderline case, however. The serum total T_4 was not markedly elevated because of the low serum thyroid binding globulin as reflected by the resin uptake test and FTI.

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References may be obtained from the author.

HYPOTHYROIDISM IN COMA FROM STATUS EPILEPTICUS PRECIPITATED BY PERSISTENT HYPONATREMIA

Persistent hyponatremia causing recurrent seizures is not a common disorder. The case presented describes a patient who repeatedly sought admission for seizure disorder and who showed hyponatremia correctable by water restriction. During the final hospitalization, the patient was in status epilepticus and died of anoxic brain damage. Lack of evidence of abnormalities on neurological examination, normal brain scans, and EEG suggest that persistent hyponatremia is causative for seizure disorder. The thyroid function studies were in the hypothyroid range. Treatment with l-thyroxine did not improve the coma suggesting that the hypothyroidism was the result rather than the cause of coma.

Case Report

A 61-year-old white male was admitted for seizure disorder on September 12, 1976, with a four-year history of seizures. Borderline hypertension was not treated by diuretics. He had been hospitalized on several occasions for habitual excessive drinking.

General physical examination showed a patient in coma, but was otherwise unremarkable. Routine laboratory studies were normal except serum sodium which showed consistently low values during his several hospitalizations *(Table I)*. Serum cortisol, skull series, brain scan, spinal fluid analysis, and EEG were normal. Syndrome of inappropriate antidiuretic hormone (SIADH) secretion was entertained, and with fluid restriction, serum sodium returned to normal range. Fifteen days after hospitalization the patient became hypothermic. The possibility of hypothyroidism as a cause for coma was considered. Thyroid studies revealed values in the hypothyroid range. Substitution of thyroid hormone did not dispel his coma or hyponatremia. His seizures were difficult to control, and he remained in coma, suggesting the possibility of anoxic brain damage. The patient was maintained on a respirator but died six weeks later. Autopsy showed evidence of severe anoxic changes in the brain but no other significant abnormalities in any of the organs. The pituitary and thyroid were normal in size and histology.

Discussion

Hyponatremia is well recognized as a cause of metabolic encephalopathy leading to delirium, seizures, and coma. The

TABLE I.
SERUM SODIUM, POTASSIUM, AND SERUM AND URINE OSMOLARITY

Date	*Serum Sodium*	*Serum Potassium*	*Serum Osmolarity*	*Urine Osmolarity*
2/11/76	126	3.8	239	399
5/5/76	112	3.2	228	566
6/21/76	121	3.0	251	641
9/13/76	115	3.7	237	565

TABLE II
THYROID FUNCTION STUDIES

	9/17/76	9/30/76*
TSH (N—below 10 μIU/ml	4.1 μIU/ml	4.0 μIU/ml
T_3RIA (N—80-220ng/ml)	<20ng/100ml	71ng/100ml
T_4RIA (N—45-130ng/ml)	6ng/ml	61ng/100ml
FT_4 Index (N—4.4-15.7)	1.4	
Plasma Cortisol (N—50-180ng/ml 8AM) (N—30-100ng/ml 4PM)	166 (8AM)	

* After treatment with thyroxine.

literature reveals rare instances of acute reversible hemiparesis caused by hyponatremia.[1]

The case presented has several interesting features. Hyponatremia was noted during several previous hospitalizations for seizures. The most common cause of hypotonic hyponatremia in a patient with normal total body sodium is the syndrome of inappropriate secretion of ADH (SIADH).[2] Nonosmolar stimuli to pituitary ADH release include emotional stress, pain, hypovolemia, CNS disorders, intrathoracic infections such as pulmonary tuberculosis or lung abscess, and a variety of drugs — including general anesthetics, narcotics, barbiturates, vincristine, clofibrate, the tricyclic antidepressants, and chlorpropamide.[3] The syndrome may also occur as a result of ADH-like substances produced from nonpituitary sources such as oat cell carcinoma of the bronchus, carcinoma of the pancreas, duodenum, or prostate.[4] Criteria for the diagnosis of SIADH are hypotonicity of body fluids, inappropriately concentrated urine, grossly normal body sodium as evidenced by lack of edema or hypovolemia, normal renal, thyroid, and adrenal function. Hyponatremia is correctable by water restriction alone. It is important to differentiate SIADH from hyponatremia of myxoedema, anterior pituitary insufficiency and Addison's disease, for in these conditions hormonal replacement alone corrects the hyponatremia.[5] The precise cause of SIADH in our patient could not be determined.

At various times during the patient's multiple hospitalizations for recurrent seizure problem, investigation ruled out such possible causes as hypoadrenalism and hypothyroidism. However, re-investigation when the patient was deeply comatose for two weeks and when he was noted to be hypothermic showed values for thyroid function in the hypothyroid range. The significance of such findings in a patient deeply comatose due to irreversible brain damage is intriguing. Substitution of thyroid did not result in improvement of his coma, hypothermia, or hyponatremia. The role of thyroid in the control of metabolic rate in nonthyroidal disease is not well understood. Burger and others[6] found that triiodothyronine and tetraiodothyroacetic acid are decreased in acute illness, while Rudman[7, 8] reported hypothyroidism evident six to seven days after the onset of coma induced by trauma. Harvey,[9] after a study of thyroid functions in seriously ill patients, reports that the changes in thyroid status found in such patients may reflect in part a normal response to such acute stress as fever, a raised free thyroxine level, and a failing response to hypothermia.

The major problem in this case was the difficulty in controlling the hyponatremia each time the patient was discharged to an outpatient status. The final hospitalization was for status epilepticus resulting in severe anoxic brain damage leading to death. Hypothermia which developed during the course of his terminal illness brings in interesting possibilities about its pathogenesis. The case presented suggests the possibility that coma from causes other than trauma also could result in hypothyroidism, shown by low thyroid function studies, and is a result, rather than the cause, of the coma. We did not study the cyclic adenosine monophosphate level in the CSF which, as enunciated by Rudman *et al.*, could have been of prognostic significance.

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References may be obtained from the authors.

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Current COMMENT

Terminal Care Needs

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ALTHOUGH our love affair with technology in America may not be ending, many have come to accept the fact that death cannot be postponed indefinitely. Death remains the physician's adversary, but with each battle this adversary need not be fought with the same intensity.

Traditionally, the wise physician has been able to recognize that point in the course of a patient's illness when death is imminent, and he has known how to provide comfort from that time on. Many centuries ago the Roman philosopher Seneca wrote: "Death is sometimes a punishment, often a gift, and to many is a favor." The late William P. Williamson, M.D., head of Neurosurgery at the University of Kansas Medical Center, was one of the pioneers in raising the consciousness of American physicians to the care of the terminally ill, and his classic paper of 1966 is well worth reading or rereading.[1]

The physician who cares for the terminally ill must understand his own feelings about death as well as the patient's. In writing about death of young patients, Easson[2] has provided us with a good scheme of the development of understanding of death which applies equally to patients and physicians. It is summarized in *Table 1.* An excellent survey of the attitudes and practices of physicians who care for terminally ill patients was conducted in Iowa by Noyes and Travis.[3] While they offered no firm conclusions, they did summarize the responses of 1,600 Iowa physicians, noting that most physicians frequently omit life-prolonging procedures or medications in caring for the terminally ill. They would do so more often if there were better methods to increase the sharing of such decisions. However, the majority expressed opposition to change in social attitudes that would allow physicians to deliberately hasten death (euthanasia).

The care of the dying presents special challenges to the attending physician. His own feelings about death, and his skill and philosophy in treatment of pain, nausea, vomiting, anorexia, hiccoughs and other discomforts, are important, but crucial is his commitment to treat the patient rather than the dread disease.

TABLE I
DEVELOPMENT OF UNDERSTANDING OF DEATH

Age	*Understanding*
Preschool	No clear concept of the finality of death
Primary School	Beginnings of the appreciation of emotional significance of death
Teenage	Appreciate the meaning of dying, but do not accept the reality of personal death
Young Adult	Death is seen as a ravisher and a destroyer
Middle Age	Accepts, but emotionally denies death
Old Age	Accepts death

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TABLE II
CHARACTERISTICS OF CHRONIC PAIN IN TERMINAL CANCER

1. A situation not an event
2. Impossible to predict when it will end
3. Usually gets worse rather than better
4. Appears to be meaningless
5. Frequently expands to occupy the patient's whole attention, isolating him from the world around

The lay literature of our time is fulsome with articles about death and dying to the point that it seems to have replaced sex. The medical literature has been less abundant. There are, however, many good papers in the medical literature, some of which are cited in the Suggested Readings. The most recent and readable essay about death and dying is that of Robert Hudson, M.D., professor of History and Philosophy of Medicine at the University of Kansas Medical Center.[4]

In recent decades, American physicians in general have lost the art and science of caring for the dying. Presently, there is much less known about managing dying than, say, managing congestive heart failure, diabetic ketoacidosis, or bleeding esophageal varices. This is regretable and needs correction. There is a renaissance of interest in the scientific study of the care of the dying, particularly in England, and many data are now available, particularly in respect to pain control.

Pain

St. Christopher's Hospice in London, England, has become the world's leading institution for demonstration, teaching, and research in care of the dying and particularly in management of chronic pain in terminal cancer. This kind of pain is characterized in *Table II*. The control of pain from cancer is perhaps the greatest challenge in the medical management of terminal illness.

If the physician can accept the philosophy that management of the terminally ill is best accomplished by treating the patient and his distressing symptoms, largely ignoring his dread disease, then the proper priorities may be set. The goal is an alert, pain-free patient who can be maintained in this state until the actual time of death, making life enjoyable as much as possible to its very end; this is the challenge of the medical management of terminal illness.

One must not assume that all pain in the terminal cancer patient is due to the malignant process. Anemic patients are likely to have angina pectoris; people who have lost weight are likely to have bed sores on bony prominences; people on opiates are likely to severely constipated and bothered with hemorrhoids, and so forth. This list of other kinds of remediable pain is nearly endless, and each type of pain may have a simple solution.

Beyond this, not all cancer-related pain requires opiates for relief; often a drug of less potency will be sufficient. Aspirin, acetaminophen, dextropropoxyphene, and phenylbutazone in regular large doses may be sufficient to eliminate pain. For example, pain from bone metastases can often be controlled by large doses of phenylbutazone 600-800 mg/day in divided doses. Indomethacin may also be effective in this situation.

A basic principle in pain control is to give enough analgesic to take away all of the pain. The terms "dependence" and "addiction" have no relevance in the care of the terminally ill. The amount of analgesic appropriate is the amount necessary to prevent any breakthrough of pain. A corollary to this principle is that only drugs with long duration of action should be used. Meperidine (Demerol) has a duration of action of only two hours and should never be used for control of chronic pain. Morphine and hydromorphone (Dilaudid) are good drugs and may be given in adequate oral doses every four hours. The concern that a cancer patient who lives longer than expected will require ever-increasing amounts of morphine has been proven baseless. Studies conducted at St. Christopher's Hospice by Twycross[5] demonstrated that only 18 (4%) of 438 patients required more diamorphine (heroin) than 30 mg/four hrs; *i.e.*, the equivalent of 45 mg morphine orally. For the patient who requires larger amounts to abolish pain, there seems to be no reason to be stingy with opiates. A few patients may require 90-120 mg morphine orally every four hours. Obviously, the physician must be alert to side effects such as nausea and constipation; the former can usually be controlled with phenothiazine by mouth or suppositories, the latter by bulk agents and laxatives. There is no particular reason to give morphine by injection as oral administration is well tolerated and gives a longer blood level. There is no evidence that heroin is any better than morphine for pain relief; the only possible advantage of heroin is smaller dosage amounts and greater water solubility.

Pain medications in the terminally ill should be given at regular dose intervals, *e.g.*, every four hours or every three hours, and *never* PRN. The goal of treatment is abolition of pain. Pain should be abolished and not allowed to return. Narcotic orders are best written in a range rather than a stated dose amount so the nurse can give more or less, basing her

decision on patient alertness and adequacy of pain suppression, *e.g.*, morphine 15-30 mg orally every four hrs.

Beyond analgesics and opiates, there are many other methods of pain control that should be considered in selected cases. Psychological methods such as diversion and antidepressants may be useful. Tranquilizing drugs and corticosteroids are often helpful. Local heat, local cold, and fixation by splinting or other means are often applicable. Various nerve blocks and even neurosurgical procedures are appropriate in some instances. Single or several-dose courses of radiotherapy may relieve local pain from bone metastases, obviating multiple trips to radiotherapy for longer courses of treatment.

Other Distressing Symptoms

After pain, nausea and vomiting are probably the most common distressing symptoms in terminal cancer. Phenothiazines — chlorpromazine or prochlorperazine — are very useful in control of these symptoms. Obviously such problems as hypercalcemia may cause nausea and vomiting also and demand another sort of therapy. These symptoms, when caused by uremia, may be relieved by reducing the metabolic acidosis with bicarbonate. However, there would seem to be no reason to restrict protein in the diet in uremia of the terminally ill.

Anorexia is a frequent problem in terminal cancer, and modest pharmacologic doses of corticosteroids, *e.g.*, prednisone 10-20 mg/day, may be most helpful and even add a sense of well-being. Alcohol should not be forgotten for its use as an aperitif as well as for its sedative and mild eurphoric qualities. The terminally ill are no more likely to become alcoholics than they are long-term narcotic addicts.

Hiccoughs are often controlled with phenothiazines. Night sweats may be controlled with indomethacin. The itching from obstructive jaundice may be reduced by cholestyramine, corticosteroids, or antihistamines. The dyspnea of respiratory failure in terminal lung cancer or other cancer widely metastatic to the lung is often controlled by morphine, making the use of oxygen unnecessary. Even intestinal obstruction from cancer can be managed expectantly with cautious, small, frequent feedings and one good vomit every day, obviating the need for a nasogastric tube and intravenous infusions *(Table III)*.

TABLE III
DRUG MANAGEMENT OF PAIN AND DISCOMFORT IN THE TERMINALLY ILL

Symptom	*Solution*
Pain	
Moderate	Aspirin, acetaminophen, dextropropoxyphene, phenylbutazone
Severe	Oral morphine or dilaudid
Nausea and vomiting	Phenothiazines
Anorexia	Corticosteroids Alcohol
Hiccoughs	Phenothiazines
Night Sweats	Indomethacin
Itching from jaundice	Cholestyramine, corticosteroids, antihistamines
Dyspnea	Morphine

Philosophical Perspectives and Conclusion

Some years ago, Day[6] summarized the care of the terminally ill rather well: "Death cannot be denied, but its dignity can be . . . by thoughtless, underfeeling, and overscientific care. Everything that is done for the dying patient should be based on the constant awareness that, although death may be postponed sometimes dramatically . . . the master plan cannot be altered."

The major problem for the physician, as detailed previously, is the decision to care for the patient and essentially stop trying to treat the dread disease. This decision, appropriately made, may help diminish the other problems that trouble physicians while caring for the terminally ill. Denial is a common physician defense and, thus, a problem. It often results in obsession with the minutiae of disease management or ignoring the patient or the family because of a feeling of failure on the part of the physician. Anger with inability to cure terminally ill patients is often displaced to associates, patients, or the patients' families. These are not desirable situations.[7]

Pain is a highly individual perception, and it usually has more than one component: *e.g.*, physical, mental, emotional, social, spiritual. All these components need identification and treatment as summarized by Saunders.[8] In this regard, members of the patient's family must be considered as they are suffering pain and grief as well. The skillful physician is one who can manage the family before, during, and after the death of the patient.

In considering the management of the terminally ill, one must not ignore the issue of euthanasia. There will always be some public, family, and personal pressure to keep this issue in the foreground. Always in the past, the pressure of euthanasia weighed most heavily on the physician. However, one of the great boons of the renaissance of the art of

caring for the terminally ill is that it eliminates the need for consideration of euthanasia. Most patients do not fear death as much as they do the process of dying. If the physician can promise the patient comfort to the moment of death and unswerving attention to the quality of the remaining days of life, ignoring the quantity of days, then euthanasia is no longer an issue.

The management of the terminally ill by physicians has been masterfully summarized by Dr. Cicely Saunders in two statements: "I once asked a man who knew he was dying what he looked for above all in the people around him, and he paused a moment and then he said, 'For someone to look as if they are trying to understand me.'" And, "I am sure the most important foundation stone we could have comes from the summing up of all the needs of the dying which was made for us in the Garden of Gethsemane in the simple words 'Watch with Me.'"

Self-Assessment Questions

1. Particularly useful in pain of bone metastases in cancer:
   a. morphine
   b. aspirin
   c. phenylbutazone
   d. acetaminophen
   e. Dilaudid
2. Time of life when reality of personal death may be appreciated:
   a. childhood
   b. teenage
   c. young adult
   d. middle age
   e. old age
3. Particularly useful in night sweats:
   a. corticosteroids
   b. aspirin
   c. Dilaudid
   d. cholestyramine
   e. indomethacin
4. Best drug available in the USA for oral use in severe pain of advanced cancer:
   a. heroin
   b. meperidine (Demerol)
   c. codeine
   d. phenylbutazone
   e. morphine
5. Useful for anorexia in terminal cancer:
   a. cholestyramine
   b. morphine
   c. indomethacin
   d. corticosteroids
   e. aspirin

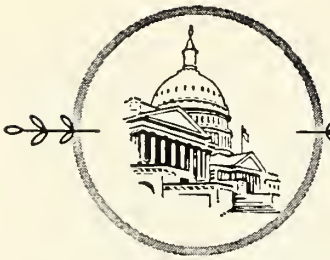
(Answers on pg. 575.)

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Socio-
ECONOMICS

Advantages of Incorporating

Fringe Benefit Programs

THERE ARE A NUMBER of benefit programs available to the incorporated professional on a tax favorable basis which are normally not available to the unincorporated physician and partnerships. These include group term life insurance, salary continuation pay plans, medical reimbursement plans, tax-free death benefits, deferred compensation agreements, dividends-received exclusion and, of course, corporate pension and profit sharing plans.

Group Term Life Insurance Benefits

Employees of professional corporations, including the stockholder-employees, may receive up to $50,000 of group term life insurance, tax free, as a fringe benefit of employment. Premiums paid by the corporation for this benefit are tax-deductible to the corporation, but are not reportable as income by the employee. Additional amounts of life insurance may be provided for the stockholder employees on a tax favorable basis.

Salary Continuation Pay Plans

Under a salary continuation pay plan the corporation may provide disability income insurance to the employees. The premiums for this benefit are deductible to the corporation and are not reportable as income by the employee. The professional should be aware, however, that benefits received while disabled, in excess of $100/week, are taxable as ordinary income.

Further information may be obtained from Midwest Pension Planners, Mr. John E. Lyons, Plan Consultant, 1010 Merchants National Bank Bldg., Topeka, KS 66612.

Medical Reimbursement Plans

With a medical reimbursement plan the corporation may provide health insurance benefits and may reimburse an employee for medical expenses not covered by health insurance. The expense of a medical reimbursement program may be deducted by the corporation, and is not normally reportable as compensation by the employees. Items that may generally be covered by this plan would include: health insurance premiums, eye glasses and contact lenses, dental expenses, and most other medical care expenses not covered by health insurance. It is also important to note that these benefits are not only available to the employees but may also be provided to the professional's family.

Tax-Free Death Benefits

The professional corporation may pay directly to the beneficiaries of the employees, up to $5,000 in death benefits. This benefit is a deductible expense to the corporation.

Deferred Compensation Agreements

This program is one of the more attractive advantages to incorporating. Through contractual agreement, a professional corporation may continue paying the salary of a retired or deceased stockholder employee. Normally these arrangements may be tied to the number of years of service in the corporation, salary level while active, and pension plans/profit-sharing plans provided by the corporation.

Deferred Compensation agreements will normally take one of the following forms:

1. The professional corporation, by contract, commits itself to pay current compensation for active employment, and a reduced amount (*i.e.*, 50% of current compensation) for a specified number of years following retirement from active service.

2. The professional corporation obligates itself to pay future compensation for a period of time that increases for each year of active employment. Through this arrangement the stockholder-employee may, for example, earn the right to an additional year of deferred compensation payments for each two years of active employment.

When properly designed, the deferred compensation agreement can postpone taxes on a professional's income until he receives it at retirement. However, such contracts will also postpone the deduction for the corporation until the income is actually paid out.

One additional advantage to the deferred compensation agreement is that it does not require IRS approval, and may be established for the shareholder-employee without consideration of other employees.

Dividends-Received Exclusion

The dividends-received exclusion provides that professional corporations owning stock in other domestic corporations may receive an 85 per cent deduction for dividends paid to it by that corporation. Since most professional corporations will normally be in the 20 per cent tax bracket, the effective tax rate would be 3 per cent on all dividends received from other domestic corporations. Care must be exercised, however, in utilizing this arrangement to prevent the professional corporation from being declared a personal-holding company by the IRS.

Medical School Tuition Loans

This benefit is particularly attractive to the professional who has a son or daughter interested in pursuing a medical career. Under this arrangement, the professional corporation executes a loan program whereby it will reimburse the medical student for his or her medical school expenses at a reasonable rate of interest (8-9%). The loan is then forgiven by the corporation for service by the student upon graduation (*i.e.*, one year of tuition is forgiven for each two years of service). The loan will then be treated as income to the new physician, who in the earlier years in practice is in a low tax bracket.

The real advantage in this benefit lies in the tax savings to the parent. The professional corporation which is in the 20 per cent tax bracket would only have to earn $12,500 to provide $10,000 of tuition loans, while the physician in a 50 per cent tax bracket would need to earn $20,000 to provide the same $10,000. Care should be taken in executing this agreement to insure it is not disallowed by the IRS. The terms of the loan agreement should be no more favorable than what might normally be made available to another individual who was not a child of the stockholder-employee.

Corporate Pension and Profit-Sharing Plans

As mentioned in a previous article, the most significant benefit available to the incorporated professional is the significantly higher contributions he may make to a corporate pension or profit-sharing plan. While the unincorporated physician is limited to contributing 15 per cent of compensation with a maximum of $7,500, the stockholder-employee is permitted contributions of 25 per cent of compensation up to $30,000.

As can be seen, the benefit programs an incorporated professional can provide for himself and his employees are numerous and substantial, and should be the prime motivator in making the decision whether or not to incorporate a practice.

Terminal Care Needs

(Continued from page 573)

Answers to Questions

1. c
2. d
3. e
4. e
5. d

NEW MEMBERS

The JOURNAL *takes this opportunity to welcome these new members into the Kansas Medical Society.*

Thomas Whitney Alderson, M.D.
1400 Polk
Great Bend, KS 67530

Larry Rotert, M.D.
301 Medical Plaza Bldg.
Topeka, KS 66604

Kenneth G. Kassebaum, M.D.
3420 E. Douglas
Wichita, KS 67208

Jay Stephen Schukman, M.D.
2601 Jackson
Great Bend, KS 67530

Robert T. Manning, M.D.
2221 N. Hillside
Wichita, KS 67219

Charles E. Wilds, M.D.
3817 E. Sixth
Topeka, KS 66607

The President's Message



What began as a pleasant dream seems to be turning into a disturbing nightmare that will not disappear upon awakening! The Physician Assistant Program, based at Wichita State University, opened in 1973 amid hopes for a method of helping the physicians in those areas of Kansas that were short of doctors, by providing persons with limited training to perform some of the more routine tasks under his supervision. The intent was both noble and timely in view of the local and national concern about the "doctor shortage" (soon to be replaced by governmental predictions of a "doctor surplus" and each "new physician generating $250,000 in health care costs annually").

The program has accepted and trained many fine, intelligent, dedicated, "blue-chip" applicants. However, although the intent was to place them in rural areas, 70 per cent or more are in urban areas. The rural doctors are not seeking help to be able to see more patients, but help in the form of another doctor to provide some relief so that he might be able to attend meetings to satisfy CME requirements, to spend time with the family, or recreational activities. He cannot legally or ethically leave his patients under the care of a PA, but must provide someone of equal training or experience in his absence.

The guidelines of the PA program were flexible and broad to allow the physician to use his best judgment in utilizing the PA. The physician was to employ no more than 2 PAs, so that they would have adequate supervision in view of their limited training. However, there have been abuses of the PAs:

Independent practice under questionable supervision with geographic separation from the physician-sponsor;

Independent practice in the same office;

Seeing hospitalized patients in the absence of their physician-sponsor;

Open criticism of their physician, implying their knowledge is equal or superior to their sponsor;

Attempting to set up offices without having an MD sponsor;

Making diagnoses and instituting therapy without checking with their physician-sponsor;

Writing prescriptions.

The Board of Healing Arts is also distressed at the turn of events. While they are charged with registration of the Physician Assistants, a recent ruling held that they had to register a PA who took the exam but failed it, because the law stated that they merely had to take the test to be registered — not that they had to pass it! Their enforcement is hampered by inadequate staff and inadequate finances.

Some hospitals have been deluged with demands for hospital privileges for the PA by the PAs and their sponsors. The hospital, because of its legal liability, separate and distinct from the MD, must stand fast in resisting those attempts. They have every right to limit or eliminate PA participation. The PA was intended to help the doctor in his office.

The Physician Assistant, on the other hand, has some honest complaints. Highly motivated and dedicated, he/she is left with the feeling that the PA is neither "fish nor foul." The role is ill-defined; duties vary from doctor to doctor because of the loosely drawn guidelines or better, non-guidelines. The PA is frustrated at seeing Nurse Clinicians operating in areas where he sees himself "competent" and "qualified." The PA senses MD and nurse antagonism and resents it as any of us would under similar circumstances.

Who is to blame? That's not the question, unless we wish to spend time and money in fruitless name calling, finger pointing, and further alienation. The problem calls for solutions, and the Medical Society should take the lead. It should meet with representatives of the PA organization so that the problems may be identified. Discussions to promote communica-

(Continued on page 578)



What's in a Name?

Unless he confounds us before publication time by surfacing in some new role, Dr. Peter Bourne has seemingly retired to a relative limbo to ponder the fickle ways of public life. It is to his credit that his transition from advising the President about drugs to doing whatever it is that limbists do (beside write books) was accomplished with dispatch and a minimum of the public discourse that is usually associated with the enforced exodus of valued presidential advisers. The departure was adequately attended by the media with (perhaps unintentional) emphasis added by their concomitant attention to the diminishing presidential image (which undoubtedly contributed to the speed with which Dr. Bourne's resignation was offered and accepted).

Dr. Bourne's transgression was relatively mild. Undoubtedly numerous persons in and out of public service — and no small number of physicians — sin as greatly every day and it seems a classical case of the error lying in being discovered. It seems certain that he had no thought that his action, though it stretched propriety, posed such a threat either to himself or the administration. The unhappy result was, in fact, due to the addition of two unexpected catalysts. First, the individual who sought to have the fateful prescription filled was not the one for whom it was intended — though that would hardly seem to matter since the one named in the prescription was consciously fictitious. But then the dissemblance was compounded by the chance presence at the scene of a representative of the law — and the chuckling demons had their day. Dr. Bourne maintained a commendable dignity by his emphatic declaration that he had done nothing illegal or unethical. While this interpretation might raise an eyebrow, the question is now moot since he has departed the scene and it has been decided not to press charges in the case.

It is an interesting irony that Dr. Bourne's position in the administration could result in his quasi-voluntary banishment on the one hand and yet be instrumental in the decision not to press charges — a case of involuntary plea bargaining. But stripped of the unique features produced by Dr. Bourne's relationship to the President — had it involved Doc Doakes of Occumucca Flats, for example — the matter would undoubtedly have led to more rigorous legal action.

So what was intended as a gesture of compassion at its inception failed in its purpose of hidden identity, brought an abrupt end (or interruption, at least) to the administrative services of an experienced individual — controversial, perhaps, but that's a prime requirement for admission to the Washington scene — and brought reverberations through the White House halls as implications of drug usage by the staff as well as other governmental minions emerged. That last point brought the inevitable grumpy reaction from the outlands that they figured that all the time, else how to explain some of the things that went on.

The real message is that Dr. Bourne apparently — and surprisingly — didn't take into account the greatest risk and the most important parameter of judgment there — politics. He might well have survived the legal and ethical tarnish of the episode, but the political sin of embarrassing his boss (whose embarrassments have almost reached the automatic flush level) was unforgiveable. In Dr. Bourne's defense, the President loyally described him as the world's leading authority on drug usage, but it must be presumed that he has now added something to his store of knowledge.

It isn't given to most of us to function in an area of such political sensitivity, but for us this serves to emphasize that an action of relatively minor signifi-

cance could develop such an impact not through its illegal or unethical features but through the political hazard. Legality and ethics are presumably fundamental characteristics acting to guarantee the worth and integrity and reproducibility of a procedure, but in practice the compromising wheel of political expedience determines the course. This matter originated as a gesture of personal politics, an accommodation for a friend, and there is probably no physician who has not performed an equivalent act. The use of the fictitious name was intended to divert the association from the personality of that friend. Thus cloaked, the prescription could provide transient relief and displace the problem to another time and place where it could be handled more appropriately — and all would be well, a successful exercise of the personal political relationship. But the very act endowed the matter with an illicit character which, on exposure, transfers it to the realm of social politics where explanation and justification are impotent.

So the conflict is not so much between the legal-ethical concept and politics as between the politics of individual relationship and the political pressures embodied in public relationships. This is about as far as it would go except for the fact that politics is intruding increasingly into the individual relationships, and the forecast is for more. This is the essence of complaint against the changing pattern of medical care, whether through nationalization of medical practice, government subsidy of various practice forms, defining and reaching a medically underserved population, or whatever — the increasing use of political considerations as a testing method. Individual patients may or may not want changes in the method of health care delivery, but the efforts toward change are inevitably political.

Nowhere is this phenomenon of a basically personal relationship being transposed to the public level with the inevitable transfer of judgment from the personal to the political level more clearly seen than in our two prime examples of governmental medicine, Medicare and Medicaid. The medical profession didn't suddenly culture a breed of grafting, avaricious practitioners to meet the needs of these programs as has become the standard reference of the media. As this significant portion of medical care has been brought into the public scope, it has become measured in statistics and volume and impersonal superficial appearances which ignore the personal services represented. It seems inherent in such programs that these personal benefits rendered to the individual patient by an individual physician are obscured in the attitude that that's the way it is supposed to be, while the gross inequities and insufficiencies of the system are attributed to the malfeasance of the practitioners.

Purists may insist that the elements of politics in Dr. Bourne's case and the examples cited differ, but we contend any difference is in fine detail, not the generic pattern. And we can expect increasingly that medical function will be tested in regard to political impact. There is progressively less in both spirit and substance that is used even in the most personal of medical relationships that is not supplied by political blessing or will be measured by political response.

And we never did find out if the girl got her Quaalude. — *D.E.G.*

The President's Message

(Continued from page 576)

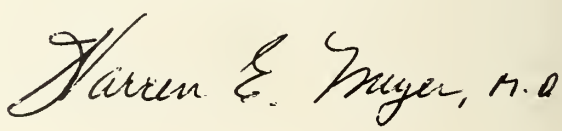
tion, understanding, and solutions can be sought to promote and protect the health of the people of Kansas.

A liaison committee of the Medical Society and the PA organization has been appointed under the chairmanship of D. Cramer Reed, M.D. You will be hearing more of this project as time goes on.

Goals should be as follows:

- An independent and unbiased review of the Physician Assistant Program to see if it is a viable program in view of the changing legislative emphasis on retaining doctors for Kansas communities as exemplified by the recently enacted scholarship program;
- Definition and purpose of the program with reasoned and appropriate guidelines;
- Defining supervision methods in keeping with the best interests of the citizens of Kansas;
- Prevention of any chance for abuse of privileges of PAs by the PA or MD;
- Providing the Board of Healing Arts with the necessary finances, facilities, and staff to do their supervision and enforcement;
- Applying just, fair, and equal treatment for those found guilty of abusing those privileges.

Fraternally yours,



President

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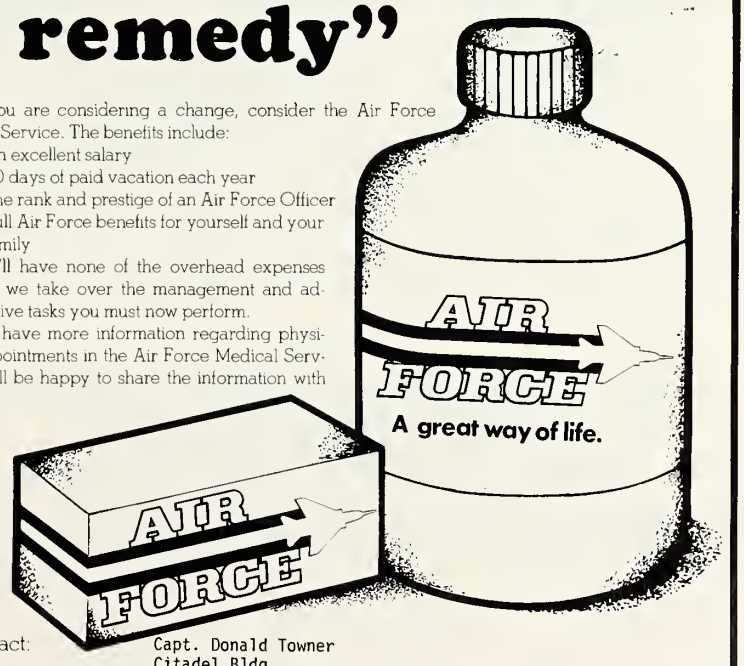
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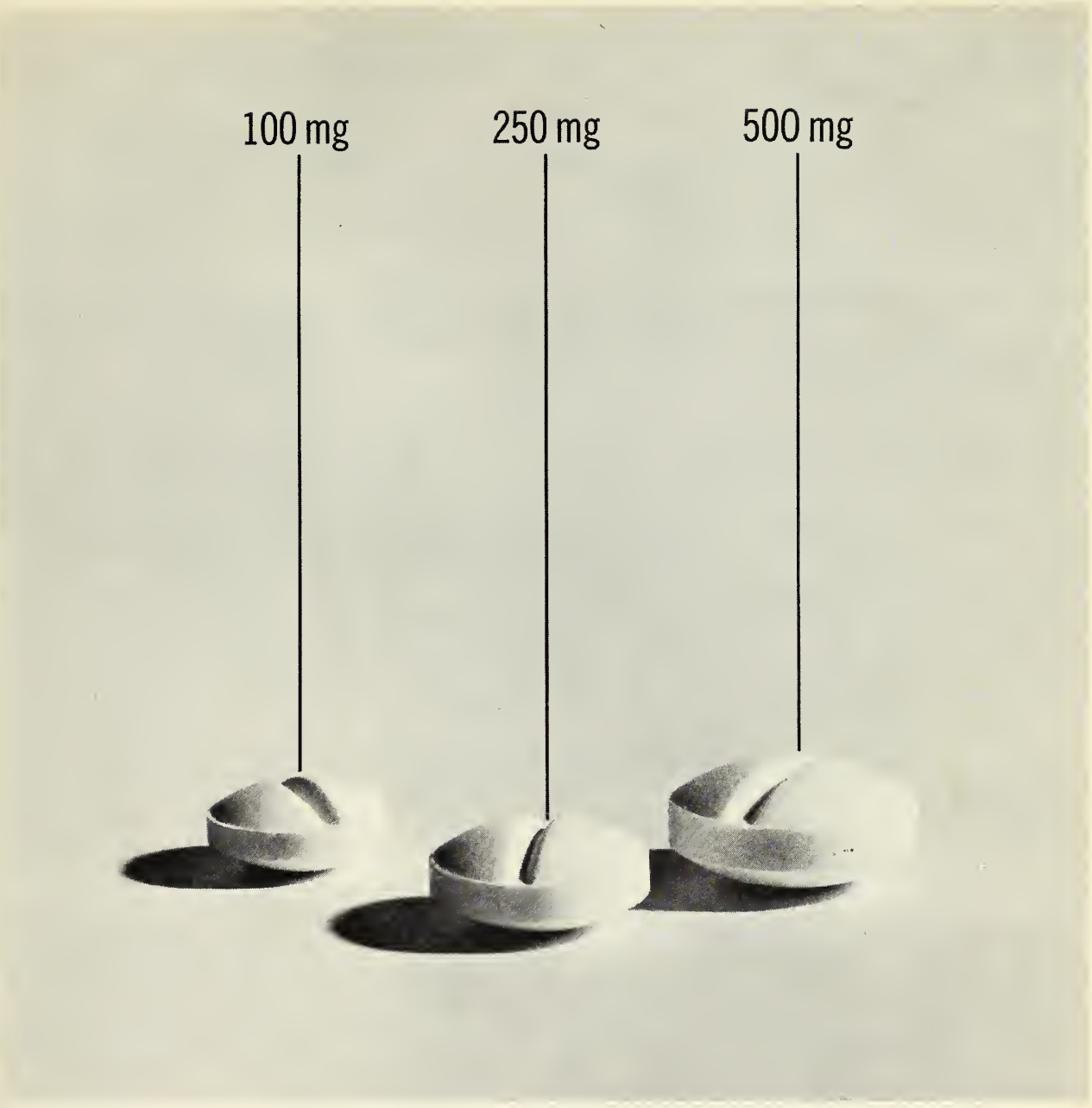
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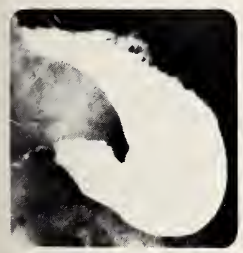
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. . . Bentyl produced definite relaxation in 8 of 10 patients. The sodium chloride produced relaxation in only 3 of 10. No side effects occurred in either group of patients.



Pylorospasm has almost totally blocked passage of barium meal.



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†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:
King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

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Brief Summary
INOICATIONS
For use as adjunctive therapy in the treatment of peptic ulcer.
IT SHOULD BE NOTEO AT THIS POINT IN TIME THAT THERE IS A LACK OF CONCURRENCE AS TO THE VALUE OF ANTICHOLINERGICS/ANTISPASMODICS IN THE TREATMENT OF GASTRIC ULCER. IT HAS NOT BEEN SHOWN CONCLUSIVELY WHETHER ANTICHOLINERGIC/ANTISPASMODIC ORUGS AID IN THE HEALING OF A PEPTIC ULCER, OECREASE THE RATE OF RECURRENCES, OR PREVENT COMPLICATION

Based on a review of this drug by the National Academy of Sciences–National Research Council and/or other information, FOA has classified the following indications as "probably" effective.

May also be useful in the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis, acute enterocolitis, and functional gastrointestinal disorders); and in neurogenic bowel disturbances (including the splenic flexure syndrome and neurogenic colon).

THESE FUNCTIONAL DISORDERS ARE OFTEN RELIEVEO BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS

For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis), paralytic ileus, intestinal atony of the elderly or debilitated patient, unstable cardiovascular status in acute hemorrhage; severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis. WARNINGS: In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. PRECAUTIONS: Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with: autonomic neuropathy; hepatic or renal disease; ulcerative colitis–Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon; hyperthyroidism; coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension; hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

It should be noted that the use of anticholinergic/antispasmodic drugs in the treatment of gastric ulcer may produce a delay in gastric emptying time and may complicate such therapy (antral stasis). Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. ADVERSE REACTIONS: Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention; blurred vision and tachycardia; palpitations; mydriasis; cycloplegia; increased ocular tension; loss of taste; headache; nervousness; drowsiness; weakness; dizziness; insomnia; nausea; vomiting; impotence; suppression of lactation; constipation; bloated feeling; severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. DOSAGE AND AOMINISTRATION: Dosage must be adjusted to individual patient's needs.

Usual Dosage: Bentyl 10 mg. capsule and syrup: *Adults*: 1 or 2 capsules or teaspoontuls syrup three or four times daily. *Children*: 1 capsule or teaspoonful syrup three or four times daily. *Infants*: ½ teaspoonful syrup three or four times daily. (May be diluted with equal volume of water.) Bentyl 20 mg.: *Adults*: 1 tablet three or four times daily. Bentyl Injection: *Adults*: 2 ml. (20 mg.) every four to six hours intramuscularly only. NOT FOR INTRAVENOUS USE. MANAGEMENT OF OVERDOSE: The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine® (bethanecol chloride USP) should be used.

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Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

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A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

Health and Safety Tip

From the American Medical Association

535 North Dearborn Street/Chicago, Illinois 60610

Carelessness Blamed In Hunting Accidents

Gun Safety Rules Given

Some millions of American males—along with more than a few females — are taking to the woods this fall, firearms in hand, in search of live targets.

These are the hunters among us.

The American Medical Association has a note of caution for those who are planning a fall safari into the fields and woods in search of birds, small animals, deer and other game.

Before you pull the trigger, know what you're firing at.

The hunter who blazes away at everything that moves is a highly dangerous animal. His quarry might well be another hunter.

Each year several hundred hunters return from fall outings via hearse. Thousands of others return with a load of bird shot or a rifle bullet to be dug out. Behind almost every hunting accident is one cause —carelessness.



Never cross over or through a fence or climb a tree with a loaded gun. It might fall and discharge. Unload first and reload after crossing. Don't shoot at a hard, flat surface. Glancing bullets can carry long distances.

Even a small bore rifle has considerable range. Know where your bullet will stop before pulling the trigger.

Keep guns away from children. Never leave a weapon unattended without unloading it. Store guns and ammunition beyond reach of youngsters, preferably under lock and key.

Always carry a gun so that you can control the direction of the muzzle if you stumble. Be sure the barrel is clear of mud, snow or other obstructions. A clogged barrel may burst. Keep the safety catch on until ready to shoot.

Leave your liquor bottle at camp, and wait until you're through the day's hunting and the guns are unloaded to take that first drink.

There is a special hazard for rabbit hunters. This is rabbit fever — tularemia. It is a serious illness. Many wild rabbits are infected. One rule of thumb is—if the rabbit doesn't run briskly when he is flushed, leave him alone. The bunny who lopes slowly along, or stands and waits, likely is sick.

October, 1978

Frank Chappell
Science News Editor
AMA

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Just one tablet b.i.d. for 10 to 14 days

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- Contraindicated during pregnancy and the nursing period. During therapy, maintain adequate fluid intake; perform CBC's and urinalyses with microscopic examination.

Before prescribing, please consult complete product information, a summary of which follows:

Indications and Usage: For the treatment of urinary tract infections due to susceptible strains of the following organisms: *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis, Proteus vulgaris, Proteus morganii.* **It is recommended that initial episodes of uncomplicated urinary tract infections be treated with a single effective antibacterial agent rather than the combination.** *Note:* The increasing frequency of resistant organisms limits the usefulness of all antibacterials, especially in these urinary tract infections.

Also for the treatment of documented *Pneumocystis carinii* pneumonitis. To date, this drug has been tested only in patients 9 months to 16 years of age who were immunosuppressed by cancer therapy.

The recommended quantitative disc susceptibility method (*Federal Register, 37*:20527-20529, 1972) may be used to estimate bacterial susceptibility to Bactrim. A laboratory report of "Susceptible to trimethoprim-sulfamethoxazole" indicates an infection likely to respond to Bactrim therapy. If infection is confined to the urine, "Intermediate susceptibility" also indicates a likely response. "Resistant" indicates that response is unlikely.

Contraindications: Hypersensitivity to trimethoprim or sulfonamides; pregnancy; nursing mothers; infants less than two months of age.

Warnings: Deaths from hypersensitivity reactions, agranulocytosis, aplastic anemia and other blood dyscrasias have been associated with sulfonamides. Experience with trimethoprim is much more limited but occasional interference with hematopoiesis has been reported as well as an increased incidence of thrombopenia with purpura in elderly patients on certain diuretics, primarily thiazides. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Frequent CBC's are recommended; therapy should be discontinued if a significantly reduced count of any formed blood element is noted.

Precautions: Use cautiously in patients with impaired renal or hepatic function, possible folate deficiency, severe allergy or bronchial asthma. In patients with glucose-6-phosphate dehydrogenase deficiency, hemolysis, frequently dose-related, may occur. During therapy, maintain adequate fluid intake and perform frequent urinalyses, with careful microscopic examination, and renal function tests, particularly where there is impaired renal function.

Adverse Reactions: All major reactions to sulfonamides and trimethoprim are included, even if not reported with Bactrim. *Blood dyscrasias:* Agranulocytosis, aplastic anemia, megaloblastic anemia, thrombopenia, leukopenia, hemolytic anemia, purpura, hypoprothrombinemia and methemoglobinemia. *Allergic reactions:* Erythema multiforme, Stevens-Johnson syndrome, generalized skin eruptions, epidermal necrolysis, urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, photosensitization, arthralgia and allergic myocarditis. *Gastrointestinal reactions:* Glossitis, stomatitis, nausea, emesis, abdominal pains, hepatitis, diarrhea and pancreatitis. *CNS reactions:* Headache, peripheral neuritis, mental depression, convulsions, ataxia, hallucinations, tinnitus, vertigo, insomnia, apathy, fatigue, muscle weakness and nervousness. *Miscellaneous reactions:* Drug fever, chills, toxic nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon. Due to certain chemical similarities to some goitrogens, diuretics (acetazolamide, thiazides) and oral hypoglycemic agents, sulfonamides have caused rare instances of goiter production, diuresis and hypoglycemia in patients; cross-sensitivity with these agents may exist. In rats, long-term therapy with sulfonamides has produced thyroid malignancies.

Dosage: Not recommended for infants less than two months of age.

Urinary Tract Infections: Usual adult dosage—1 DS tablet (double strength), 2 tablets (single strength) or 4 teasp. (20 ml) b.i.d. for 10-14 days.

Recommended dosage for children—8 mg/kg trimethoprim and 40 mg/kg sulfamethoxazole per 24 hours, in two divided doses for 10 days. A guide follows:

Children two months of age or older:

Weight		Dose—every 12 hours	
lbs	kgs	Teaspoonfuls	Tablets
20	9	1 teasp. (5 ml)	½ tablet
40	18	2 teasp. (10 ml)	1 tablet
60	27	3 teasp. (15 ml)	1½ tablets
80	36	4 teasp. (20 ml)	2 tablets or 1 DS tablet

For patients with renal impairment:

Creatinine Clearance (ml/min)	Recommended Dosage Regimen
Above 30	Usual standard regimen
15-30	½ the usual regimen
Below 15	Use not recommended

Pneumocystis carinii pneumonitis: Recommended dosage: 20 mg/kg trimethoprim and 100 mg/kg sulfamethoxazole per 24 hours in equal doses every 6 hours for 14 days. See complete product information for suggested children's dosage table.

Supplied: *Double Strength (DS) tablets,* each containing 160 mg trimethoprim and 800 mg sulfamethoxazole, bottles of 100; Tel-E-Dose® packages of 100. *Tablets,* each containing 80 mg trimethoprim and 400 mg sulfamethoxazole—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 40, available singly and in trays of 10. *Oral suspension,* containing in each teaspoonful (5 ml) the equivalent of 40 mg trimethoprim and 200 mg sulfamethoxazole, fruit-licorice flavored—bottles of 16 oz (1 pint).

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Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

Please see back cover.

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Please see reverse side for summary of product information.

IMPAIRED PHYSICIAN

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NO. XI

Volume LXXIX, Number 11, November, 1978

The JOURNAL of the KANSAS MEDICAL SOCIETY

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral—Adults:* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d. Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

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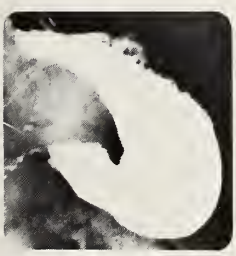
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†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:
King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

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Brief Summary
INDICATIONS
For use as adjunctive therapy in the treatment of peptic ulcer.
IT SHOULD BE NOTED AT THIS POINT IN TIME THAT THERE IS A LACK OF CONCURRENCE AS TO THE VALUE OF ANTICHOLINERGICS/ANTISPASMODICS IN THE TREATMENT OF GASTRIC ULCER. IT HAS NOT BEEN SHOWN CONCLUSIVELY WHETHER ANTICHOLINERGIC/ANTISPASMODIC DRUGS AID IN THE HEALING OF A PEPTIC ULCER, DECREASE THE RATE OF RECURRENCES, OR PREVENT COMPLICATION

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the following indications as "probably" effective.

May also be useful in the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis, acute enterocolitis, and functional gastrointestinal disorders); and in neurogenic bowel disturbances (including the splenic flexure syndrome and neurogenic colon).

THESE FUNCTIONAL DISORDERS ARE OFTEN RELIEVED BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS.

For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation

CONTRAINDICATIONS. Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis); paralytic ileus, intestinal atony of the elderly or debilitated patient, unstable cardiovascular status in acute hemorrhage, severe ulcerative colitis; toxic megacolon complicating ulcerative colitis, myasthenia gravis. WARNINGS: In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. PRECAUTIONS: Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with: autonomic neuropathy; hepatic or renal disease; ulcerative colitis—Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon, hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension; hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

It should be noted that the use of anticholinergic/antispasmodic drugs in the treatment of gastric ulcer may produce a delay in gastric emptying time and may complicate such therapy (antral stasis). Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. ADVERSE REACTIONS: Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia, urinary hesitancy and retention; blurred vision and tachycardia, palpitations, mydriasis, cycloplegia, increased ocular tension; loss of taste; headache, nervousness; drowsiness; weakness, dizziness, insomnia; nausea; vomiting, impotence; suppression of lactation; constipation, bloated feeling; severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. DOSAGE AND ADMINISTRATION: Dosage must be adjusted to individual patient's needs.

Usual Dosage: Bentyl 10 mg. capsule and syrup: *Adults*: 1 or 2 capsules or teaspoonfuls syrup three or four times daily. *Children*: 1 capsule or teaspoonful syrup three or four times daily. *Infants*: ½ teaspoonful syrup three or four times daily. (May be diluted with equal volume of water.) Bentyl 20 mg.: *Adults*: 1 tablet three or four times daily. Bentyl Injection: *Adults*: 2 ml (20 mg.) every four to six hours intramuscularly only. NOT FOR INTRAVENOUS USE. MANAGEMENT OF OVERDOSE: The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine® (bethanecol chloride USP) should be used.

Product Information as of October, 1976

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Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

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Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

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All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

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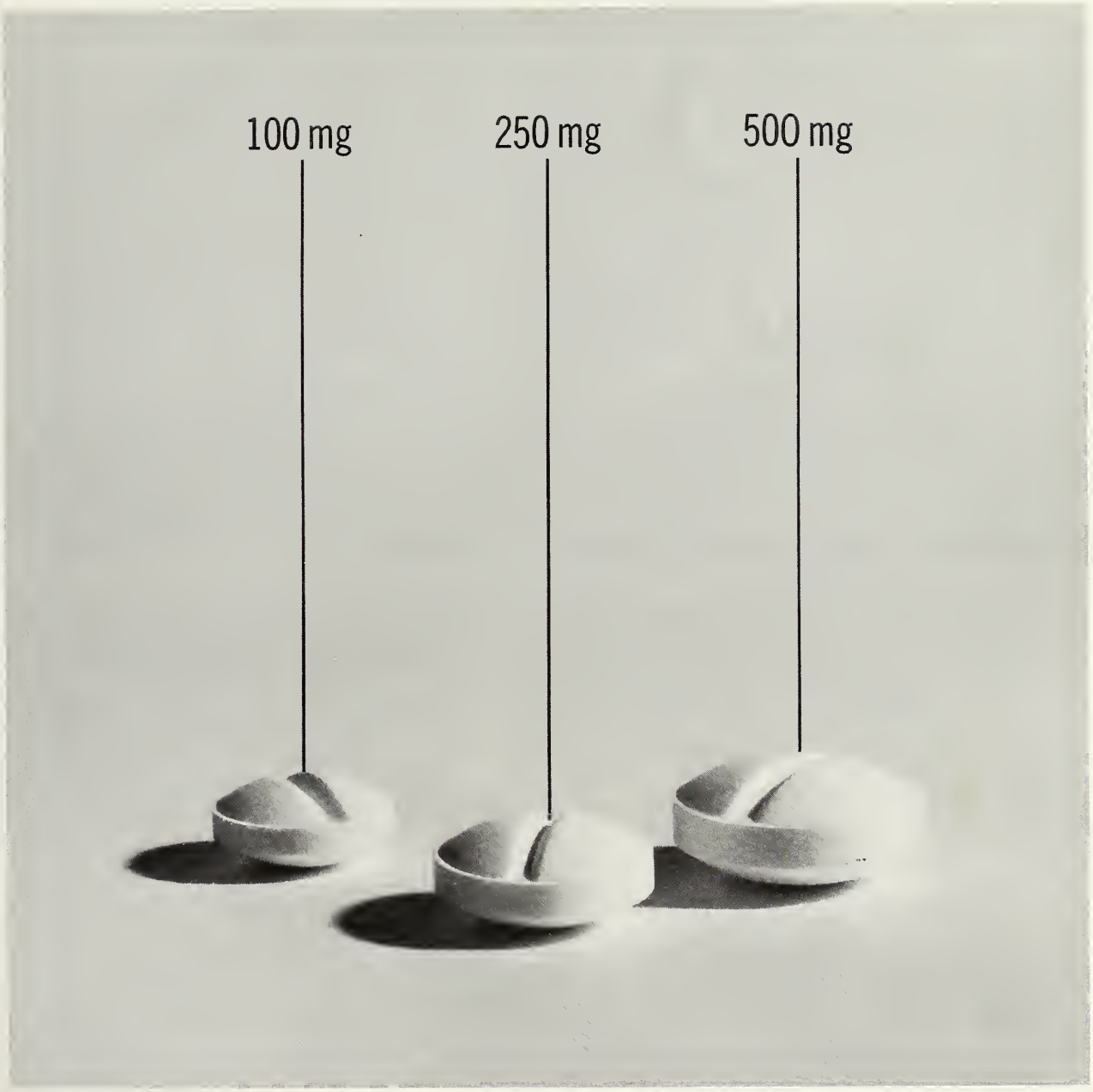
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David D. Crane, M.D.
146 Morningside
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Eleanor P. DeMoss, M.D.
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Physician Distribution

One of the thornier problems confronting health care planners, policymakers and providers — the geographic distribution of health manpower, particularly physicians — was the subject of a recently completed study by the General Accounting Office.

In a report to Congress the GAO said that $430 million in Federal funds had been spent during fiscal years 1972-77 in an effort to increase the supply of physicians in shortage areas.

According to the GAO, some 384,900 non-Federal physicians — 369,800 MDs and 15,100 DOs — are unevenly distributed throughout the United States. Of the MDs, approximately 292,200 (79%) are providing patient care. But although this works out to 137 physicians for each 100,000 persons, a disproportionately large number are located in urban areas, where the physician-to-population ratio is significantly higher than in rural areas. Furthermore, the GAO reported, distribution is also "inequitable" within urban centers.

As for the 20,000 new physicians on whom data were available, they followed the path of their predecessors: 90 per cent of the 1967-71 U. S. and foreign medical graduates licensed located in cities.

The study attributed the urban concentration of recent graduates primarily to the fact that most physicians, other than general and family practitioners, choose urban practice locations. The latter are more uniformly distributed between urban and rural areas, the GAO said.

"Preference for rural or urban living and the availability of clinical support facilities and personnel were equally important factors affecting the location decisions of a sample (1,066) of physicians licensed in 1971," the GAO found. "As a group, however, professional considerations, including clinical support, contact with other physicians, and continuing education opportunities, played the largest role. Overall, the physicians stated they had not been greatly influenced by economic factors, such as income potential and the availability of loans."

Reporting on the National Health Service Corps, the GAO told Congress:

"The Corps has undoubtedly increased the availability of physicians' services in communities designated by HEW as having a critical shortage of health manpower. However:

- "The Corps has not adequately considered the demand for medical services when assessing the need for physicians in shortage areas. This, coupled with its decision to place a minimum of two physicians at most sites to the extent possible, has resulted in many physicians being underused in terms of patients served at sites in operation one year or longer.
- "The Corps has experienced difficulty in recruiting physicians willing to voluntarily practice in the more remote, less populated areas. Consequently, many of these sites have remained unstaffed for periods ranging up to 4 years.
- "Corps officials look to the HEW scholarship program with its shortage area service obligation to provide a sufficient supply of physicians in the future. But, because of deferments for graduate medical education, HEW officials do not expect a substantial number of physicians to be available until fiscal year 1979.

"It should be recognized, moreover, that the number of physicians authorized by the Corps for its unstaffed sites may exceed the number needed as evidenced by the low use of Corps physicians at many sites in operation one year or longer. This raises serious questions to GAO concerning the extent of unmet demand for health care in some of these areas and, therefore, the number of additional physicians with scholarship service obligations that will be needed to serve in HEW-designated shortage areas.

- "From inception through July 1976, only 42 physicians — out of a total of about 800 who served in the Corps — remained in the shortage areas or were planning to do so, as private practitioners, which is a major program objective."

As for loan repayment programs, the GAO found them relatively ineffective.

"As of October 31, 1977," the report said, "The Federal loan repayment program attracted only 762 physicians (about 1.7% of those eligible) to shortage areas in return for loan repayment. Moreover, the majority of those who participated through February 1976 probably would have established practices in those shortage areas anyway. Thus, it seems the program provided financial benefits predominately to physicians who already had decided on shortage area practice."

The GAO found it hard to assess the effectiveness of area health education centers.

"The long-term nature of the program, the lack of clearly defined national strategy, and different developmental stages and program strategies among the 11 area health education centers make identifying and assessing the program's impact difficult. Nevertheless, GAO believes this program conceptually has considerable long-term potential to indi-

rectly improve health manpower distribution by overcoming some of the important professional objections to shortage area practice."

Both the preceptorship and family medicine training programs were too new at the time of GAO's review to determine their impact on increasing the supply of physicians in shortage areas.

In an effort to assess state and private efforts to improve access to primary care medical services in rural areas through programs relying heavily on nonphysician providers (including nurses and physician extenders) the GAO visited four such programs:

The North Carolina Rural Health Centers; Kentucky Frontier Nursing Service; Checkerboard Area Health System; and East Kentucky Health Services Center. All use physician extenders as principal providers of health care in rural clinics. The physician extenders provide services under the supervision of physicians, but physicians are not always present when the services are performed.

The inability to receive reimbursement from Medicare (part B) and some other third-party payers for physician extender services at independent sites such as those discussed above had apparently restricted their potential widespread use.

"In 1965," the GAO reported, "when Medicare was enacted, there were few if any physician extenders working and no allowance was made for their reimbursement." But, the report continued, "on December 13, 1977, the President signed into law the "Rural Health Clinic Services Act," authorizing reimbursement under Medicare (part B) and Medicaid for services rendered in certain rural health clinics in underserved areas. Among the services covered are those of physician assistants and nurse practitioners, whether or not a physician is physically present at the time the service is provided.

"The act also requires the Secretary of HEW to conduct demonstration projects in urban medically underserved areas with respect to reimbursement on a cost basis for services provided by physician-directed clinics which employ physician assistants and nurse practitioners.

"In GAO's view, now that reimbursement for physician extender services rendered in rural clinics in underserved areas has been authorized, projects which rely extensively on physician extenders at satellite clinics or in mobile units with backup from physicians in larger neighboring communities could constitute an approach for providing health care to communities in the Nation otherwise unable to attract or retain physicians."

The General Accounting Office recommended that the Secretary of Health, Education, and Welfare should:

1. Develop guidelines for assessing under what circumstances it would be appropriate to assign health care providers to entities requesting Corps assistance and the number and type of provider(s) that would be most appropriate;

2. Require communities and other entities requesting Corps health care providers to conduct studies which identify, to the extent possible, the number and types of residents located therein who are likely to seek care from a Corps-sponsored practice;

3. Develop multiyear projections to assess the total number of physicians with scholarship commitments that will be needed to serve in shortage areas;

4. Make an analysis of the extent to which family practitioners and other specialists are locating in HEW-designated shortage areas and, based upon this analysis, submit to the Congress recommendations for financially supporting those programs which constitute the greatest resource for providing health care to medically underserved areas;

5. Work with the states to identify those areas having health manpower distribution problems and develop a strategy for marshaling resources — federal, state, and private — to establish an integrated program designed to provide health services in the manner most appropriate to each area;

6. Examine those programs which rely on physician extenders to help deliver health services to those areas otherwise unable to attract physicians and consider seeking legislation which would provide federal funds to help develop those programs found to be most useful.

Addressing its recommendations to Congress, the GAO said it believes "it is doubtful that a separate loan repayment program is still needed to attract physicians to shortage areas in view of: (1) expanded Corps scholarship program and number of physicians expected to be available for shortage area service; and (2) discretion available to the Secretary of HEW under the Health Professions Educational Assistance Act of 1976 to repay the newly authorized federally insured health professions student loans.

"Therefore, the Congress should reconsider whether the loan repayment program for physicians needs to be continued since

- "it has not induced substantial numbers of physicians to enter shortage area practice and
- "many physician participants apparently re-

(Continued on page 620)



Auxiliary News

Statewide workshops were conducted in Manhattan and Dodge City for all interested auxiliary members during the month of October. Those attending were responsive and enthusiastic about the programs being offered this year. The requests for the traveling exhibit, "Learning Center on Body Pollution" for fifth and sixth graders are so numerous that it will be difficult to meet the demand, and we are considering the advisability of constructing another traveling center. There has been a great deal of interest in promoting the SCORE program, our health education program for teenagers. Hopefully, we will see it off the ground soon.

Response to the questionnaire on Enrichment in Medical Marriage, sent to Auxiliary members, has been most enthusiastic. As the spouse of a physician for over thirty years, I agree with other auxilians who believe that medical marriages are different. Physicians possess some rare and fine qualities of dedication, caring, and giving that make them the fine physicians they are. I realize I am prejudiced, but I believe they also tend to choose marriage partners who are somewhat special. Each phase in our lives presents a different challenge and a different set of problems — the stress and strain of medical school, internship and residencies, the transition from the academic world to that of the practice of medicine, the addition of children, the loneliness of moving to a new and sometimes different community, the lack of privacy in some situations and, for us, the sharing of our physician spouses with so many others. These adjustments are not always easy. Some of us weather the storm and others do not. We don't pretend to have all the answers but we are willing to learn and to help. The questionnaire is completely anonymous but will serve as a guideline in our attempt to develop programs for the future. We are already receiving requests and suggestions from Auxiliary members for programs on marriage enrichment and programs geared to the needs of the spouse of the medical student, intern, and resident physician.

These are the areas in which the Auxiliary has a very unique place in its ability to serve. I sincerely urge you to support your spouse as we endeavor to fill some of the needs in these areas.

A meeting of our Executive Board and Long Range Planning Committee was the beginning of a review of our total Auxiliary program in an effort to make us more efficient and more effective.

Again, I urge you to support your spouse in her Auxiliary efforts, and if she is not a member encourage her to join us. We can do more together.

Sincerely,
Mrs. Wm. H. Crouch (Jean)
President, KMS Auxiliary

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Final classification of the less-than-effective indications requires further investigation.

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The Impaired Physician Program of The Kansas Medical Society

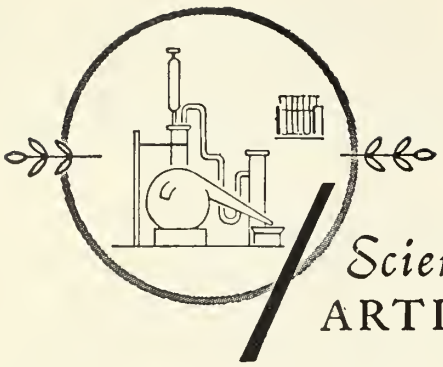
All facets of the physician's constitution — genetic, environmental, mental, educational, social, and ultimately, professional — conspire to create an image of strength, self-sufficiency, and independence. Unless the physician is able to consciously implement coping mechanisms and construct channels of personal interdependence, he/she is exceptionally vulnerable to the intense pressures inherent in medical practice today. The response to such pressures is often impairment — impairment frequently manifest through depression and chemical addiction. Unchecked this may result in inadequate or improper care for the patient and incapacitation or suicide for the physician.

The same perspectives that contribute to this vulnerability also make it virtually impossible for the physician to visualize, acknowledge, and reverse impairment without help. Therefore, the profession has accepted the responsibility for recognition of impairment and intervention at the depression/addiction level. The goal of the Impaired Physician Program of the Kansas Medical Society is to assist the impaired physician in achieving maximum effectiveness, both personally and professionally, with a minimum of disruption in his/her practice of medicine. Members of the committee have established a mechanism designed to attain this goal.

The purpose of the program is described in detail in the August (roster) issue of the *Journal*. Its resources are available to any KMS member with any impairment to effective practice of medicine. It can be activated by the physician, his professional peers, his family, a patient, or anyone with reason to believe the physician is in need of help. All physicians will be accorded respect and understanding, and all information will be treated confidentially.

To contact the committee, call:

(913) 235-2383



Some Considerations

Depression and the Healing Process

GEORGE M. PENN, M.D., * *Topeka*

PARADOXICALLY, in the oldest myths of healing, it was precisely because the healer was wounded that he had the power to heal.[1] In other words, the capacity of the physician to experience himself simultaneously as healer and wounded, as physician and patient, enhances his ability to collaborate in the healing process; by accepting and integrating both of these aspects of himself, he enables his patient to experience the healing aspects of himself.

Unfortunately, the physician all too often can only identify with the healing aspect of this health-sickness polarity and projects his inner wounds and illnesses onto the patient — thus he is trapped by his own omnipotence. His patient, in turn, remains estranged from his inner healer, having projected this aspect of himself onto the physician. If the relationship remains like this — that is, both doctor and patient maintaining these unconscious projections upon one another — movement toward cure may be impeded or even stymied.[1]

Changing this relationship which reinforces the health-sickness polarity is easier said than done. There are a number of patients for whom the perception of themselves as sick provides some benefit to their state of adaptation or well-being. Unless the physician is aware of this and is able to consider the meaning of the illness for his patient in the context of his patient's current life situation, no movement toward real cure occurs, despite physical and psychological interventions.

The physician's effectiveness as healer is influenced by his own attitudes toward himself. Self-examination facilitates awareness of the experience of being human, thus maximizing the physician's ability to be simultaneously healer and human.

Moreover, for the physician, being the healer can also provide great adaptive value. This value is not readily perceived unless the physician is willing to examine as honestly as he can what he may gain from this incomplete image of himself — that is, his perception of himself as healer but not as wounded. Feifel (1975) has noted that some physicians have greater than average fears of illness and death. Going into medicine is an intellectual way of denying — but at the same time trying to manipulate — these realities. Hence the need of these physicians to cure is very great. This self-image helps to avoid seeing the shadowy, wounded side of themselves, as well as the periphery of their own limitations. Yet when those intractable cases finally come, the physician in this condition opens himself up to the full onslaught of his patient's illness, as well as being overtaken by his own that he has never fully acknowledged.[1]

* Section Chief, C. F. Menninger Memorial Hospital; Chairman, KMS Impaired Physician Program Committee.

Address reprint requests to Dr. Penn, P.O. Box 829, The Menninger Foundation, Topeka, KS 66601.

Regrettably, little of this is taught in medical school. Few physicians have had any formal experience in talking about and sharing with one another the emotional stress of medical practice. Medical education tends, rather, to reinforce the role of physician as "magical healer" — the one who must always be strong and perfect, the one upon whom others may always lean, the one who must always live up to the demanding ethic to do good and to do all that he can.

This destructive health-sickness polarization must be changed first by admitting its existence and then by beginning to produce a professional world of peer support and sharing rather than competition, of openness rather than secrecy, and of cooperation rather than isolation.[2] Failure to do so makes it virtually impossible for us to meet the conflicting demands and needs of patients, family and community, as well as our perfectionistic expectations of ourselves; furthermore, failure to acknowledge our own inner wounds and vulnerabilities contributes to the strikingly high rate of depression, suicide, alcoholism and other substance abuse rates in the medical profession, and provides a continuing source of marital conflict.

Dr. Will Menninger once made the remark that it is not necessary to get sick in order to get better, and indeed it is not; but it is necessary to be more realistic about ourselves and about our assets and liabilities if we are to get better without first having to get sicker.

Socrates told us long before Freud that the unexamined life is not worth living. Unless we can see and accept ourselves as we are, we cannot hope to make wise or useful decisions about our own destiny, nor can we hope to more fully understand the ills of our patients. Too often, as physicians, we live our lives from hour to hour, avoiding intimate relationships, keeping too busy to reflect on the choices and commitments that we have made or on the direction in which our lives are moving. Moreover, it is extraordinarily difficult for us to look at ourselves closely and honestly. Most of us never even try until we are forced to do so by a crisis, a major life change or some blunt confrontation by family, friends, or peers — in other words, until we become sicker.

Even when we do make an honest effort, the image that we see of ourselves is often distorted by projection, by denial, and by other adaptive/defensive tendencies which are present in all of us and which operate to make reality conform to our own beliefs and expectations so that we see what we need to see, hear what we need to hear, and believe what we need to believe.

Self-examination in the form of several questions provoking reflection upon the psychological and interpersonal aspects of your own life may be painful, but may also give a clearer picture of who you really are, what you stand for, and toward what you are headed. Such self-awareness will not only have a healing effect on you, but can also increase your sensitivity to the adaptive value of illness in many of your patients as you are able to consider more empathically the effect of psychological and interpersonal determinants as they play a role in the presenting problems of your patients.

Question 1:

What are your goals in life?

How realistic are they, and how well do they truly reflect your values and priorities? Most of us spend an enormous amount of time in our professional lives in planning and setting goals. It is likely, however, that we put very little effort into personal planning on our own behalf. Even the most reflective among us rarely asks this question, except in moments of crisis or despair, or in the middle or later years of life when we begin to wonder just exactly what our life has meant and what we are going to do with our remaining years.

Rosen (1976) has used the term *destination sickness* to describe the gnawing sense of dissatisfaction and impatience, feelings of being boxed in, and doubts about choice of profession and very lifestyles that physicians experience during mid-career crisis.[3] Has it all been worth it, we ask? The "failure" of success may be summed up in an old Indian proverb: "Beware of what you set your heart upon; you might obtain it."

Question 2:

Does your use of your time and energy truly reflect your priorities?

Try a simple but enlightening experiment. Find some time for quiet reflection and list on a piece of paper what you think is most important to you in priority order. Keep a diary for a week or longer, recording just how you actually spend your time. Transcribe the results to your priority list; you will probably be surprised to note some glaring discrepancies. At some level, outside of our awareness, we are likely to perceive an inconsistency between what we stand for, believe in and desire, and what actually is.

Question 3:

How courageous are you?

Consider whether you have the courage and the

willingness to look at yourself honestly and to judge yourself fairly — acknowledging your assets, while at the same time acknowledging and owning your shortcomings and learning to deal with losses.

A prayer by Reinhold Niebuhr expresses this kind of courage quite well:

> "God grant me the serenity to accept the things I cannot change, the courage to change the things I can, and the wisdom to know the difference."

Question 4:

What is the quality of your personal relationships?

How do you balance your need for contact with your need for privacy? How do you deal with issues of psychological closeness? Are you so afraid of hurting others, or of being hurt yourself that you never really get involved?

Too often relationships are seen as self-perpetuating, and as a result they are treated in a casual way and often not cared for with even the same solicitude that we devote to the maintenance of our own automobile or lawnmower.

Perhaps none of us has a truly mature relationship, for that is an ideal that encompasses so many important aspects. It has been said that a mature relationship must be based upon respect; it must contain open, candid, and honest communication; the content of that communication must be intimate in the sense of sharing our innermost feelings and concerns truthfully. To develop this sort of full rewarding and mature relationship takes time — time together and time by oneself.

Question 5:

What is the meaning of love in your life?

There are many definitions of the word love. However, if we think of love as caring, it becomes clear that the opposite of love is not hate; rather it is indifference or not caring. To live a life without loving and being loved is to live a life of emptiness.

All of these questions have one thing in common — they deal with relationships and they imply that in our relationships with people as well as with things, our responsibilities go beyond mere adaptation. We must try to change our world as well as to accept it. But it is not enough to change our environments to meet our capacities and needs; we must change ourselves in order to exist in the new environments we have created. This is the dilemma we all face — to accept ourselves and our world as well as to change both, and to find stability within change.

We all remember the biblical commandment: "Love thy neighbor as thyself." Only one part of that commandment tells you to love your neighbor; the other part tells you to love yourself. Freud and the Bible meet in the insight that you cannot love your neighbor unless you love yourself. Self-deprecation and self-hatred are the root cause of sin as well as crime, of misery as well as cruelty and illness.

Self-understanding, self-criticism and self-acceptance, in the context of our goals and objectives, are what these questions are all about. They imply relationships to self and to others in complex and interactive ways. If we cannot or will not gratify our own needs, we cannot provide gratification to others. If we have not fully experienced pain or sorrow, we cannot understand or empathize with these feelings in others. There is a line in a musical play, "The Fantasticks": "Without the hurt the heart is hollow."[4] Philosophers as well as psychologists have been trying to tell us these things in various ways. Shakespeare, as always, puts it simply and elegantly: "To thine own self be true, and it must follow as the night the day, thou canst not then be false to any man."[5]

In summary, depression is not only an illness, it is part of the human condition. We all experience it, some of us more than others. The core experience of depression is that of emptiness, or an inner hollowness reflecting a lack of relatedness to others and to someone greater than ourselves — a higher power, God. It is this lack of relatedness, manifesting itself in various ways, that lies at the heart of what has been called the "anguish or sadness of the soul."[3]

A scene from a Thornton Wilder play based upon the fifth chapter, seventh verse of the Book of John, captures the essence of depression and the healing process. In this scene, an angel is speaking with a physician who has appeared at the pool wherein miracles occur to get a cure for his depression. The angel says to the physician: "Without your wound where would your power be? It is your very remorse that makes your low voice tremble into the hearts of men. The very angels themselves cannot persuade the wretched and blundering children on earth as can one human being broken on the wheels of living . . . in love's service only the wounded soldiers can serve — draw back, this moment is not for you."

What Wilder knew was that the relating of one human being to another demanded only one thing, and that was human experience. One need not be a physician to bring this to a sick suffering human being — all you need be is a human being.

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The Alcoholic Physician

An Overview

JOHN C. CONNELLY, M.D.,* *Topeka*

ALCOHOLISM is no respector of persons. You will find this illness among physicians just as surely as you will find it among the unemployed and the unskilled. The old stereotypes of the skid row bum, or the terminally-ill cirrhotic patient, have been found to represent a very small percentage of the total alcoholic population.

In the manual on alcoholism recently published by the American Medical Association, the following definition is offered: "Alcoholism is an illness characterized by significant impairment that is directly associated with persistent and excessive use of alcohol. Impairment may involve physiological, psychological or social dysfunction."[1] The critical word in this definition is the word "persistent." The alcoholic person continues to drink daily or periodically despite growing problems that are related to the drinking.

Until recently the issue of the alcoholic physician was neither confronted nor even recognized by most medical societies. If medical societies were reluctant to confront this problem, laymen were even more reluctant to think of their doctors as possibly having alcohol problems or other chemical dependence problems.

In the past few years the situation has changed rather dramatically. State medical journals now devote whole issues to the topic of the impaired physician;[2] seminars and workshops on this subject are well attended; and in the lay press, alcoholism, as one of the potential problems for physicians, is openly discussed.[3] For the most part, the new perspective seems to be that doctors are not immune to problems and that these problems need to be recognized and treated. The extent of this change in perspective is particularly well exemplified in an article written by a middle-aged board certified surgeon about his own alcoholism and published in a widely circulated journal.[4] This physician not only uses his own name, but concludes by saying, "In short, life has taken on such new dimensions for me since recovery that I can almost say, I am glad I am an alcoholic."

Alcoholism in the physician is discussed from several perspectives: the nature of alcoholism, characteristics of the alcoholic physician, and considerations in treatment.

The Nature of Alcoholism

Alcoholism in the United States has long been recognized as a major health problem. The National Institute of Alcohol Abuse and Alcoholism (NIAAA) estimates that alcohol is used by 70 per cent of the adult population, and approximately 10 per cent of this group have or will develop serious alcohol related problems. The ravages of this illness are seen not only in the broken lives and disrupted families of the alcoholic person but also in the staggering annual cost to the nation in terms of absenteeism, lost production, motor vehicle accidents, and medical expenses.[5]

There is no single cause of alcoholism; psychological, sociological, physiological, and possibly genetic factors are all thought to play a role. Alcoholics cannot be recognized by personality type. Psychotic patients may become alcoholic as may neurotic and borderline patients, even patients with apparently well integrated personalities. It is true, however, that because of alcohol's ability to reduce tension and pain, to provide oral gratification, to temporarily "dissolve" a harsh conscience, and to give a greater sense of relatedness, persons who are impulsive and alienated from others, with low frustration tolerance and punitive superego, are particularly vulnerable.

It is estimated that the life expectancy of the alcoholic person is 12 years less than for the general population.[6] This is due in part to the high incidence of suicide in alcoholics, quoted as being anywhere from 5-20 per cent.[7-9] Excessive mortality can also be related to the high incidence of accidents such as car wrecks, fires, and drownings. Serious health problems — such as cirrhosis of the liver, pancreatitis, and alcoholic heart disease — round out the

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general causes of premature death in the alcoholic person. Deaths related to alcoholism are particularly tragic because of the potentially treatable nature of this illness.

Characteristics of the Alcoholic Physician

Whether physicians are more prone to alcoholism and other forms of drug dependence than the general population remains unclear. It is often said that addiction problems are occupational hazards of the physician. Statistics on the incidence of alcoholism are quite speculative, but the American Medical Association[10] estimates that approximately 6 per cent of physicians are significantly impaired by alcoholism/drug addiction or mental illness. Of this impaired group, perhaps as many as 40 per cent are alcoholic, either solely or in conjunction with other drug abuse or depression. An incidence of this magnitude would not be higher than that postulated by the NIAAA for the general population; nevertheless, a conservative estimate of the number of physician addicts in the United States would be 20,000.

Studies are appearing in the literature that discuss the characteristics of physician addicts, but only two studies dealing specifically with the alcoholic physician will be mentioned here.

In Murray's[11] study of physicians admitted to psychiatric hospitals in Scotland and England, 39 per cent were alcoholic with 6 per cent of this group being psychiatrists and 33 per cent surgeons. Almost all of the physicians diagnosed as alcoholic also abused other drugs. Pre-alcoholic careers ranged from repeated failure to spectacular success. Physicians in Murray's sample rarely sought help primarily on their own accord, and treatment was carried out in an inpatient setting. The results of treatment were disappointing at the time of a five-year follow-up when only about 30 per cent of the physician group were practicing satisfactorily.

On the other hand, Bissell and Jones[12] gathered data on 98 recovering alcoholic physicians who had been abstinent for at least one year. The sample was gathered primarily through the organization, International Doctors in Alcoholics Anonymous. In Bissell's sample, the breakdown according to specialty was roughly the same as the national breakdown according to specialty, except for an overrepresentation of psychiatrists (17%). Fifty-seven per cent had been addicted only to alcohol; the remainder to alcohol plus at least one other drug. Over one-third in Bissell's group of recovering alcoholic physicians felt their psychiatrists did not accept alcoholism as a serious problem in its own right, and this had been an obstacle to recovery. Forty-seven per cent had been arrested at least once, and this was a considerably higher percentage than those who had been disciplined by medical societies or hospital staffs. Bissell concludes that the public is still much more aggressive in identifying alcohol problems in the physician than are medical peers.

In our series of 31 physicians treated over the past three years in the Menninger Clinic Alcoholism Recovery Program, 35 per cent abused only alcohol; 42 per cent, alcohol plus at least one other drug; and 23 per cent, other drugs with no alcohol abuse. Similar to Murray's experience, surgeons were overrepresented in our sample, accounting for nearly 30 per cent of admissions, while Ob/Gyn specialists accounted for 26 per cent.

In terms of age distribution, one peak was noted between the ages of 30 and 40, and a second peak between 50 and 65. In the first group, alcohol or drug abuse usually was present before the development of a successful medical practice, and often had started prior to medical school. This group tended to have more severe psychopathology — often in the borderline range. In the 50-65 age group, successful practices had generally been established well before the onset of alcoholism, and the development of problems was usually associated with mid-life crisis or depression.

In only 15 per cent of the Menninger sample was treatment primarily at the patient's initiative; 37 per cent were under strong family pressure; and 41 per cent under strong work related pressure to seek treatment.

Vaillant[13] has written about the vulnerability in physicians to the development of alcoholism or drug dependency. In a prospective study including 47 physicians he found that, compared with socioeconomically matched controls, physicians were much more likely to have unstable marriages and to use alcohol and drugs heavily. Although these difficulties are often assumed to be occupational hazards, Vaillant found that their presence or absence was strongly correlated with adjustment prior to medical school. Only the physicians with the least stable childhood and adolescence appeared particularly vulnerable. In them, patient care seemed to be an attempt to give something to others — in an emotional sense — that they had not received themselves.

Considerations in Treatment

In regard to treatment, the physician-alcoholic must be thought of first as a person with alcoholism, and second as a physician: he may be in the early stages of alcoholism or in the late stages; he may or

may not have other serious problems. He deserves the same kind of careful diagnosis and treatment as any other patient; special arrangements because he is a physician can only work to his disadvantage.

Talbott[14] has identified certain job-related factors in the medical profession that must be kept in mind and brought into the treatment process:

1. The physician has available to him all kinds of potent mood-altering drugs in addition to alcohol, and more often than not alcohol is not the only drug being misused;

2. The demands on a physician's time are great and so are the tensions of the job, and alcohol and other drugs can provide an enticing way to temporarily relax tensions without sacrificing time;

3. A patient wants to think of his doctor as an omnipotent figure, and this is a very appealing and flattering role for the physician. The price that he pays, however, is a denial of his own needs, problems and limitations, at least on a conscious level. Alcohol provides some gratification of infantile needs without the necessity to acknowledge these needs;

4. The physician's self-esteem may be closely tied to his role as a provider of help; therefore, to become a patient may be experienced as very threatening to self-esteem;

5. Physicians are often high achievement-oriented individuals with well developed obsessive-compulsive personality styles. Frequent characteristics of such personality styles are dissatisfaction with accomplishments and difficulty in feeling comfortable playing just for the fun of it.

Alcoholism in the physician is treatable, and many resources are available to the physician and his family to facilitate the recovery process. First, however, the problem must be identified. This will generally need to be done by others because the alcoholic physician uses the same denial as other alcoholic persons, probably even more intensely. Once the problem has been identified, the physician must be motivated to deal with it, and the earlier he gets into treatment, the better his chances for recovery. Oftentimes, the major source of motivation for treatment comes from the imminent threat of either marital dissolution or professional censure. For the physician, a threat to his practice of medicine is probably the most powerful motivating force. This is where impaired physicians' programs can play an important role. More and more states are enacting such programs, and although they vary from state to state, they have certain things in common. The goal is to help the alcoholic physician or other impaired physician recognize his impairment and accept treatment before his problems pose an overwhelming threat to his medical practice. Only if the physician refuses to seek treatment and continues to deteriorate will his medical license be in jeopardy.

Hospital treatment of the physician alcoholic is the treatment of choice for several reasons:

1. The emotional process of becoming a patient (*i.e.*, one who is dependent on others for help) is a difficult one because it goes against the physician's role and self-image, but no change can occur without it;

2. Detoxification is safer and more manageable in a hospital setting. Physician-alcoholics, more than most others, are prone to use additional mood-altering drugs intermittently, and drug combinations can significantly complicate the detoxification process;

3. Hospitalization provides a respite from the pressures and frictions of home and job; it allows the physician an opportunity to reassess priorities in his life and develop a renewed sense of hope that things can be different;

4. Hospitalization for the physician also provides a respite for his family and enables them to become involved more easily in the process of re-establishing a more functional total family system;

5. Physicians generally do not take easily to Alcoholics Anonymous, but AA provides as tremendous a source of ongoing support for the physician as for other people. In a hospital treatment setting, intensive exposure to AA and its philosophy can be accomplished;

6. Careful long-term planning can best be accomplished in a hospital setting based on an in-depth assessment of physical, psychological, social, and spiritual factors. Because recovery from alcoholism cannot be completed during a brief six-week hospital stay, additional therapeutic involvement is usually necessary. This generally involves some form of psychotherapy in addition to Alcoholics Anonymous participation. Sometimes a longer period of hospitalization is needed as well.

Summary

Physicians are not immune to alcoholism although the incidence is probably no higher for them than for the general population. Alcoholism is a treatable illness, and the physician generally brings significant resources to the treatment process in the form of involved family members, history of previous achievement, and financial resources. At the same time he brings some notable resistances — including the use of his own intellect in the service of denial. Hospital treatment is the treatment of choice, not

only to more effectively confront denial, but also to remove the physician from external pressures and allow him the opportunity to reassess priorities and establish with others an ongoing rational treatment plan. Physicians can and do recover from alcoholism but first they must become patients.

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Some Considerations

(Continued from page 600)

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The M.D.eity

Some Personality Vulnerabilities of Physicians

JOHN CODY, M.D.,* *Hays*

THE PSYCHIATRIC RESIDENT'S first male patient — following a year's training on the Women's Section — turned out to be a severely depressed, board certified psychiatrist. Not surprisingly, there were problems. First, he knew more about psychiatry than the resident. Second, the resident had trouble thinking of him as a patient and, as a result, found himself drifting out of the therapist's role and into that of the colleague. Third, the patient unwillfully intimidated the ward staff, and as a consequence they were unable to limit his autonomy appropriately or see that he followed the course of treatment prescribed. And fourth, it was difficult for everyone to gauge how severely incapacitated he was because of his intelligence, air of importance, and denial that he had any weaknesses or that there were any problems in his life that he could not handle himself.

In his inexperience, the resident concluded that the problem was more than half his; he simply did not know enough to treat a man whose knowledge of the field so greatly exceeded his own. He also concluded that the rest of the difficulty must reside in the patient. He had a set of defenses and resistances to the inroads of psychotherapy that were, it appeared, unique to him in their impenetrability. A considerable number of physician-patients later, this psychiatrist has learned that he overrated both the hampering effect of inexperience and the uniqueness of the patient's defenses. For he was a physician (and that is saying a lot), while this author was the resident.

Physicians, indeed, are very likely to possess a personality structure that one might claim (with room, of course, for numerous exceptions) is characteristic of the profession. It is a way of being, a style, a stance toward the world that is the product of native endowment, life experiences, professional conditioning, and family dynamics and values. Certainly, there are great strengths woven into the structure, but also some vulnerabilities and potential stress points that may crack under pressure. The personalities of physicians often make them difficult to treat, as in the case of the psychiatrist-patient; often cause them much suffering; and in the long run may limit their lives both in depth and in duration.

Let us first consider native endowment plus life experiences. Anyone with the capacity to get through medical school has inherited high intelligence. It has been said that an IQ of at least 125 is a prerequisite if the candidate for medical training is to successfully complete the course of study. But brainpower is not enough; physiologically, considerable resilience, stamina, and energy are requisite. The frail, over-tender, sluggish, and nerveless are unlikely to be able to meet the rigorous physical and emotional demands involved in that most intensive of all initiations.

The unique structure of the physician personality, reinforced by the medical school experience, predisposes to flawed coping abilities, thus rendering the physician vulnerable to pressures that impair his effectiveness, both professionally and humanly.

These qualities of intellect and body tend to set the future physician apart long before he enters medical school, and this very setting apart encourages the development of a self-image deviating from the average. Typically, he finds himself to be the brightest student in his grammar school and among the brightest in high school. The same pattern usually continues to a great extent in college also, considerably reinforcing the future physician's image of himself as a superior person which, indeed, in some ways he is.

It is axiomatic that in the struggle for life's prizes one uses whatever means one possesses to obtain an edge over the competition. The future physician learns very early that his brains and energy are two very considerable assets, and that by making the most of them he can attain at least his share, and perhaps more than his share, of those prizes. Intellect and energy may tend then to become overvalued and overworked, sometimes at the expense of other important facets of personality — such as aesthetic and religious feeling, intuition, and human relatedness.

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The great temptation is always to put all the eggs in the single basket that has proved itself to be the handiest and most commodious. Physicians are not alone in this; the beautiful woman and the athletic man often do this too. While they have them, they use their physical attributes to influence others and to gain power. Similarly, the embryo-physician uses his intelligence and energy for that purpose. When beauty and strength fail, as they inevitably do, the once beautiful woman and the once athletic man may be left with few resources with which to cope for the remainder of life. Likewise, the person who stakes everything on his brainpower and horsepower, when he encounters a facet of life where not these but other qualities and abilities are called for, may find himself quite at a loss.

In sum, the future physician, because of his intellectual and kinetic endowment, tends to become an over-specializer; moreover, because of his academic successes, he tends to become an over-specializer with a keen sense of superiority to others. Overvaluing intellect and energy, he tends to undervalue other facets of personality, and may even look down on those whose lives derive their meaning from the concentrated play of these other facets.

When the student gets to medical school, professional conditioning is superimposed on this constellation of attitudes and values. Generally speaking, medical school is a sobering, humbling and, regrettably, sometimes humiliating experience. With a few brilliant exceptions, the student finds that he is no longer smarter than those around him because now everyone is smart. Brains and energy still count, of course — in fact they count more than ever. But for most students the best they have serves only to keep them going, and preserves them from the disaster of flunking out. It does not make them outstanding and bring down on their heads the accolades of fellow students and faculty alike, as it once may have done. Applause and the academic spotlight, therefore, are gratifications most students must learn to do without, along with the other pleasures that are necessarily given up in outfitting one's self for the demands of the profession. The student consequently often feels put upon, overworked, and underappreciated. Lacking much in the way of social life and just plain fun and entertainment, and usually living on a very limited budget, he dreams of the halcyon days after graduation when his former exceptional status will be restored. He will then become once more a superior person who gives orders instead of taking them, and whose knowledge and capability will be recognized by everyone. The godly demeanor of some physicians is in part, no doubt, a rebound from the scut work and low status of medical school. It is as if the new physician vows to himself that he will never knuckle under to anyone or anything again. Henceforth, he will be the captain of his fate, the master of his soul, and the final authority in everything that pertains to him, his environment, and his work.

Central to the medical school experience is also the gradual assumption of life and death responsibilities for patients. Ground into the student is the concept that his job is to fight pathology and conquer death — two opponents whose elemental might says much about the overwhelming and grandiose nature of the physician's ideal. Such an ideal — heroic and beautiful as it is — necessarily involves him in much falling short, many disappointments, embarrassments, and reminders of his inadequacy. To guard against depression, guilt, and failure of nerve the student — and later the physician — may tend to shut out of awareness all appreciation of his real shortcomings, and adopt an artificial self-image compounded of a sense of invulnerability and infallibility. Physicians who do this exude self-confidence and dauntless faith in their methods and skills, often to the great benefit of fearful, dispirited patients. But the price paid by the physician in exchange for this facade of omnipotence is that he begins to resemble a monument instead of a man, and in the end may be painfully lonely because no one really knows him as he is. He has prestige and admiration, but his capacity for intimacy and love may well have atrophied beyond hope of restoration.

Through excessive reliance on objective intelligence the physician is prone to size up every situation in a rational, cognitive way, leaving feelings out of the picture altogether, almost as though they were the awkward, undignified, encumbering residua of animal evolution. His marital and sexual life may suffer as a result. If, instead of treating his wife and children as special objects of his interest and devotion he treats them simply as a set of more than usually demanding patients, he abandons the role of husband and father for that of in-house sage and lawgiver. Whenever one hears a medical wife speak of her husband, not as Jim or Bill or even as "my husband" but as "The Doctor," one suspects that there are serious interpersonal difficulties in the family.

Admission policies of medical schools are partly responsible for the existence of what might be called this archtypal physician personality. A strong science background is still the most emphasized prerequisite for admission, and there is but lip service given to the relevance of those fields — literature,

painting, music, history, philosophy — that provide cultural perspective and deepen the imaginative and feeling life. As a result, the accepted students begin their medical careers with a strong bias in favor of a hard, objective, rational approach to the world and to other human beings. Nothing that happens to them in medical school is likely to alter this bias; rather, what is mostly confirmed there is the macho prejudice that the humanities — and especially the arts — are the soft, vague, muddle-headed concerns either of women or of men who are less than masculine. The "real" man — including, of course, the autocratic surgeon, the cerebral internist, and the super-cool pathologist who are the students' feared and respected models — would have no time for such fripperies. The irony inherent in this he-man psychology is that physicians often strongly identify with their mothers, and have a deep impulse to be nurturant, to take care of people — a conventionally "feminine" activity at the other extreme from machismo. But our cultural stereotypes make it difficult for men to accept such feelings in themselves. Consequently, they may deny and smother them, along with other interests, pastimes, and talents not especially endorsed by their environment. The result is that many physicians become work addicts who cannot conceive that there could be more to life than practicing medicine. They never retire, rarely leave town, turn every family vacation into an orgy of continuing education, and can think of nothing better to do on their days off than chart-work or golf.

It has been said that one cannot even hope to survive in medical school if one is not an obsessive-compulsive personality. Indeed, psychological testing of medical students does show that a large percentage possess that personality configuration. Its origins go back to childhood, before the future physician is aware of his intellectual advantages, before his school experiences, good and bad, before his medical school initiation and his rebound from that. It begins, typically, with parents who are orderly, neat, clean, punctual, conscientious, and ambitious. From the dawn of his awareness of others and the world about him, the archtype physician-in-embryo adopts these habits and attitudes as his own. He becomes a good, conforming child, so well behaved that he seems prematurely grave and wise for his age. He is also perfectionistic, overcontrolled, and may lack true spontaneity. His standards — like those of his parents — are high, and he suffers much tension and anxiety in trying to live up to them. He is stubborn, given to ruminative self-doubts, and cautious. But — and this is where he has an advantage in medical school — he is persevering in the pursuit of a goal; he is a master at suppressing his feelings and denying himself pleasure whenever his feelings and the urge for pleasure threaten to interfere with the pursuit of that goal. In extreme cases there is a driven, relentless quality to his capacity for work.

Many patients take advantage of physicians and make unreasonable demands on them. Also, as with ministers, they often expect their doctor to be more than human — more patient, warm, self-sacrificing, altruistic, and virtuous than other men. Such expectations, in addition to placing the physician under the same kind of load his parents and mentors once placed on him, also play into his own tendency to regard himself as much above others. There is danger in this attitude. As some sage once said: To try to be more than human is to risk becoming less than human.

Of course, not all physicians conform to this pattern. Some physicians are not obsessive-compulsive personalities; many do not react to schooling in the ways suggested; nor do they develop the kind of self-image portrayed. Nevertheless, the stereotype contains more than a kernel of truth. When doctors become excessively "physicianly," when our personalities grow over-rigid, when we identify too completely with our professional roles, this is the direction in which we tend to go wrong. Most physicians, however, manage not to go too far in this direction and, happily, remain responsively, realistically, and approachably human. With their intelligence, vitality, interesting life, deep experience of the joys and sufferings of their fellow man, such happy and balanced guardians of life and health can be among the wisest and most delightful of human beings. Certainly, most doctors are fine, admirable people. When one gets to know them they are not difficult to love. This author is convinced of it; he married one.

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.

Disease Processes

Chemical Addiction Is Too a Disease

ROY NEWTON NEIL, M.D., *Hays*

SEVERAL years ago the author was invited to participate in a seminar and was assigned the task of presenting alcoholism and drug addiction (hereafter referred to as chemical addiction) as disease processes. This was a number of years after the AMA had officially elevated these processes to the disease status. Unfortunately, the attitude of some health care professionals remains something akin to "let the bastards suffer because they deserve it."

In order to perceive chemical addiction as a disease process, it first becomes necessary to define disease. Webster's dictionary succinctly defines disease as "a condition in which bodily health is impaired." Health is then defined as "a state of being sound or hale in body, mind and soul" — a multidimensional definition.

For purposes of clarity, this definition of health is expanded to include four dimensions — physical health (body), social and emotional health (mind), and spiritual health (soul). A brief outline of the dimensions of health and persons in our society who influence each is as follows:

Dimensions of Health

Physical — physicians, nurses, ancillary health care providers (pillpushers)
Social — parents, schoolteachers, media (babysitters)
Emotional — psychiatrists, psychologists (skulljockeys)
Spiritual — clergymen, churches (skypilots)

Physical Health

When we think of physical health or health of the body, we often think in terms of its absence — cardiovascular disease, cancer, diabetes, cirrhosis, colds, flu; and we also think of doctors, nurses, hospitals, pills, shots, operations, pain. When people are physically sick, they expect someone to give them something or do something that will make them feel better. Hence, the awesome power of physicians — they and only they can write a prescription (the magic wand); and the influence of nurses — they often administer the medication. It is no wonder that this formidable team with its associates are regarded, often with mixed feelings, as the purveyors of physical health or, less respectfully, as pillpushers.

Social Health

Social health has to do with learning and coming to understand the give and take of family life and

Webster's dictionary defines disease as "a condition in which bodily health is impaired." The processes and ramifications of this impairment in the chemically addicted are explored and their significance emphasized.

structure, with learning the technical and social skills necessary to exist in a complex technological society; *e.g.*, if you cannot read or write or add and subtract, it is very difficult to be healthy in our society. Social health also involves comprehending the need for social order and the reasons for laws and rules to govern behavior; *e.g.*, understanding why you should not take something that does not belong to you; why evacuation of one's bowels in public streets is undesirable; and why driving under the influence of a chemical substance is and should be illegal. In our society, social health is supposed to be learned in our homes, our schools, and our streets from a variety of people referred to as babysitters. Unfortunately, we too often do not learn.

Emotional Health

Emotions are normal. Anxiety, depression, grief, anger, jealousy, love, hate, fear, and other emotions are all a part of normal living; however, there are healthy and unhealthy ways of dealing with emotions. Anger is a normal emotion — it is healthy to deal openly and directly with anger and our reasons for it, and to work through it without residual resentments or depression; it is unhealthy when angry to take a baseball bat to an old lady; to rape someone; to drive 100 mph on a public highway, or to get drunk or take a pill and do all three. Love is a normal emotion — it is healthy to love one's spouse; it is often unhealthy to love someone else's.

Contrary to prevailing opinion, we are not genetically constituted in such a fashion as to know naturally and automatically healthy ways to deal with normal emotions. How we deal with our emotions is learned behavior; and if our learned behavior is unhealthy enough, we go or get sent to psychiatrists, psychologists (skulljockeys) and their associates — hopefully for successful retraining in these skills.

Spiritual Health

In this context, spiritual health should not be construed in the classic theological sense. Rather, it has to do with coming to understand, more realistically, concepts such as blame and guilt, right and wrong, and the experience of happiness.

Blame and guilt are almost universally misused, and this misuse results in agonizing malaise. We do something that harms someone and they blame us and we feel guilty or vice versa. Used correctly, blame and guilt should imply that someone does something deliberately, intentionally, and with malice aforethought to harm someone else or himself. Most of the things that people do to harm others or themselves are not deliberate and malicious; rather, they are mistakes — mistakes of being human — done out of passion, lack of consideration, desire, selfishness, and especially, because of unhealthy expressions of emotions. Parents do not generally set out deliberately to do things that harm their children or vice versa. Although parents and children will make some decisions and do some things that will result in harm to one another, blame and guilt are not appropriate in these situations. The last person who was perfect got into a whole bunch of trouble and much of mankind still worships Him. No one has his first beer following the junior prom with the intention of becoming an alcoholic and doing a great deal of harm to himself and others — yet in this country it happens to approximately one out of ten people who drink.

I am right, so therefore, *you must be wrong.* This is another common source of spiritual impairment. Each of us has an entirely different genetic code (unless we are identical twins) that reacts in a special way with our environment so that no two people are exactly alike. Therefore, what is right and wrong for one individual will seldom, if ever, be exactly right or wrong for another individual, and that is the way it should be. The author has a fraternal twin brother — both grew up in as close to similar environmental circumstances as is humanly possible. Our genetic codes are different, our reactions to that environment are different, our values, goals, beliefs, and lifestyles are different. For one of us to say to the other, "I am right and you are wrong" would be arrogant and stupid, but we have been guilty of both. And so have members of the human race over the centuries — always heinously invoking their god to prove themselves obviously and unequivocally right and righteous and the other fellow(s) as obviously wrong and villainous. *I am right and you are wrong* — nonsense!

Another common fallacy in the area of spiritual health concerns the right to happiness. No one has any right to happiness! An all-American basketball player who is a Rhodes scholar and a millionaire's son has no more right to happiness than an achondroplastic dwarf with an IQ of 50 and born in poverty, because neither can take any personal credit or blame for what he does or does not have with regard to physical attributes, intelligence, talents, abilities, or resources. In that regard we are all equal. Our physical attributes derive from a power greater than ourselves, and spiritual health involves coming to understand that we will be happy only to the extent that we use those physical attributes — whatever they are — to be useful to the people around us, and that unhappiness results if we use them for self-gratification, self-glorification, and self-gain. We and we alone are responsible for experiencing or not experiencing happiness.

Traditionally spiritual health in our society is the responsibility of our clergymen and our churches — the skypilots who try to take us above ourselves to new heights of living.

Chemical Addiction Is a Complete Disease

Chemical addiction affects all dimensions of health. Addicts are physically unhealthy with gastric ulcers, gastritis, esophageal varices, gastrointestinal bleeding, oral-pharyngeal cancers, fatty metamorphosis, cirrhosis, hepatitis, pancreatitis, diabetes, heart disease, convulsions, hallucinosis, chronic organic brain disorders, intracranial hemorrhage, polyneuropathies, anemia, clotting disorders, malnutrition, renal disorders, lacerations, broken bones, broken heads, broken bodies, burns, ad infinitum.

Addicts are socially unhealthy with broken marriages, broken homes, and broken children. They steal things that do not belong to them, they drive under the influence of chemicals, and they evacuate their bowels in public places.

Addicts are emotionally unhealthy. They are chronically and abnormally angry, depressed, fearful, hateful, anxious, jealous, and incapable of love.

Addicts are spiritually unhealthy. They are filled with guilt; blame everyone else for their problems;

are always right and never wrong; and are certain that their physical attributes, talents and abilities are of their own making, that they have a right to happiness and that, furthermore, something or somebody has the responsibility for making them happy.

Chemical addiction is insidious, all pervasive, and one of the most complete diseases — if not the most complete disease — known to mankind. Those unable to understand the concept of chemical addiction as a disease do not belong in the health care delivery system.

Furthermore, if a person suffers from spiritual malaise, a doctor cannot give him a prescription for a pill that will make him spiritually healthy. If a person has not learned healthy ways to deal with normal emotions, a doctor cannot give him a prescription for a pill that will make him know how to deal in a healthy fashion with those emotions. If a person has not learned how to read and write or add or subtract or appreciate the need for social order, a doctor cannot give him a prescription for a pill that will make him know those things. And many doctors, out of ignorance and magnificent benevolence, write thousands of prescriptions for hundreds of thousands of pills to try to cure these problems. This results in spiritually, emotionally, and socially unhealthy people, who are also chemical abusers or addicts, and who subsequently become physically unhealthy.

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Current COMMENT

Hyperuricemia and Gout: Eminently Treatable Disorders

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GOUT is a familial metabolic disorder characterized by: (1) hyperuricemia; (2) deposition of sodium urate in and around (tophi) joints; and (3) recurrent attacks of acute monarticular or pauciarticular inflammatory arthritis. Effective treatment of acute gouty arthritis and asymptomatic hyperuricemia require quite different approaches to therapy. Acute gout is controlled with potent anti-inflammatory drugs such as indomethacin or colchicine, which typically are not uricosuric. Hyperuricemia is controlled with antihyperuricemic drugs that are not anti-inflammatory. Initiation of antihyperuricemic therapy during an acute attack of gouty arthritis may well prolong the acute episode.

Gout is one of the few rheumatic diseases for which there is a definitive laboratory test, namely, demonstration of negatively birefringent urate crystals from a joint effusion or tophus *(Table I)*. For crystal examination, joint fluid should be placed in a tube free of anticoagulant. The anticoagulant calcium oxalate mimics the appearance of urate crystals; as these are also water-soluble, an excess of an aqueous anticoagulant or diluent will dissolve them. Urate crystals may be detected in body tissues if they are fixed in absolute alcohol rather than aqueous formalin. More than one inflammed joint should be aspirated for detection of crystals if none is present in the first synovial fluid examined; not all acutely involved joints have demonstrable urate crystals. Rarely, gout and septic arthritis may coexist in the same joint; synovial fluid containing urate crystals should be cultured for aerobic and anaerobic bacteria if there is any suspicion of an articular or systemic infectious process.

Treatment of Acute Gout

Once the physician has made a definitive or presumptive diagnosis of gout *(Table I)*, he may use one of several drugs to control joint inflammation effectively *(Table II)*. For an established diagnosis, indomethacin (Indocin®) is currently the drug of choice, because of its efficacy and relative safety. Phenylbutazone is also satisfactory but should not be used in patients with marginal cardiac compensation because it promotes sodium retention and may precipitate congestive heart failure or pulmonary edema; this is much less of a problem with indomethacin. If the diagnosis is in question, the drug of choice is colchicine because few other forms of inflammatory arthritis respond to this medication. Inpatients may receive intravenous colchicine slowly in a volume of 20 ml normal saline or 5 per cent glucose in water. This technique is particularly useful for postoperative patients on parenteral fluid

TABLE I
DIAGNOSIS OF GOUT

Definite:
Demonstration of negatively birefringent urate crystals from joint effusion or tophus

Probable:
1. Male over age 40
2. Acute monoarticular or pauciarticular (2-4 joints) asymmetric arthritis in lower extremities
3. Hyperuricemia
4. Prompt response (<24 h) to colchicine (not specific for gout)

* Assistant Professor of Medicine; Director, University Arthritis Clinic.

Address reprint requests to Dr. Lindsley, Department of Medicine, UKSM-KC, 39th & Rainbow Blvd., Kansas City, KS 66103.

TABLE II
DRUG MANAGEMENT OF ACUTE GOUTY ARTHRITIS

Drug	*Dose/Route*	*Relative Contraindications*
Colchicine	0.6mg/h p.o. ≤ 12 doses	Active peptic ulcer disease, liver or renal disease
	1-2 mg IV q 6h ≤ 4 mg/24h	Leukopenia, poor veins, liver or renal disease
Indomethacin	150 mg p.o. initially then 50 mg tid with taper	Active or recurrent GI disease, psychosis, pregnancy, childhood
Phenylbutazone	200 mg p.o. tid with taper ≤ 7 days	Blood dyscrasia, CHF, childhood
ACTH	40 USP units IM tid × 2-3 days	Diabetes, adrenal insufficiency

therapy and also has the advantage of avoiding the GI toxicity, *i.e.*, diarrhea, associated with oral therapy. If extravasated during infusion, however, colchicine produces marked cutaneous irritation. Oral colchicine may also be used; unfortunately adequate relief of joint pain and inflammation is usually accompanied by the onset of drug-induced diarrhea. Other forms of inflammatory arthritis that may respond to colchicine include pseudogout (calcium pyrophosphate disease) and sarcoid arthritis. Newer nonsteroidal anti-inflammatory drugs such as ibuprofen (Motrin®) or naproxen (Naprosyn®) have been used experimentally and appear equivalent to presently approved drugs for acute gout.

All these drugs have their greatest efficacy if given early in a course of acute inflammatory arthritis. With any of these drugs, the patient should note a substantial decrease in pain and swelling within 24 hours, and virtually complete resolution of joint inflammation within three to five days.

Treatment of Intercritical Gout

Once the initial episode of acute arthritis has been treated satisfactorily, the physician has two additional concerns for this patient. One is prophylaxis of recurrent attacks of acute gouty arthritis; the second is control of the hyperuricemia which usually accompanies acute gout *(Table III)*. Prophylaxis of acute attacks is best accomplished by administration of colchicine at a dose of 0.5 or 0.6 mg one to three times daily. This is indicated if the patient is having two or more acute attacks/year. Reduction of hyperuricemia can be accomplished with drugs that either enhance the excretion of uric acid or inhibit its synthesis *(Table IV)*. Of patients with primary gout, one-third have excessive synthesis of uric acid, one-third have defective excretion, and one-third have features of both; these conclusions were drawn from patients with gout receiving radiolabeled precursors of uric acid. A 14-year epidemiologic study in Framingham, Massachusetts, indicated that patients with serum uric acid levels exceeding 9 mg/dl had an incidence of gouty arthritis of 85-90 per cent; those in the 7-9 mg/dl range, 15-25 per cent; and those less than 7 mg/dl, 1-8 per cent. In addition to patients with gout those with asymptomatic hyperuricemia, especially if greater than 9 mg/dl, may develop chronic interstitial nephritis secondary to urate deposition.

Thus, for most patients with hyperuricemia, it is appropriate to utilize drug therapy to lower the serum uric acid below its maximum plasma saturation of

TABLE III
SERUM URIC ACID VALUES

State	*Level (mg/dl)**
Hyperuricemia	>7.0-7.5
Hypouricemia	<2.0
Mean levels:	
Male	6.5
Female	5.5

* These values were obtained using an enzymatic method. Values obtained by colorimetry (autoanalyzer) typically run 0.5-1.0 mg/dl higher.

TABLE IV
ACTIONS OF ANTIHYPERURICEMIC DRUGS

I. Probenecid
  Enhances excretion of uric acid by inhibiting renal tubular resorption
II. Allopurinol
  Converted to oxypurinol by liver, has long half-life in plasma (about 28h)
  Decreases synthesis of urate
    Inhibits xanthine oxidase (i.e. blocks conversion of hypoxanthine and xanthine to uric acid)
    Inhibits purine synthesis

TABLE V
ANTIHYPERURICEMIC AGENTS

Drug	*Dose/Route*	*Relative Contraindications*
Probenecid	250-500 mg p.o. tid to qid	Overproduction of uric acid, GFR <50% of normal, salicylate administration
Allopurinol	300-600 mg p.o. daily	Liver disease, bone marrow suppression, purine analogue (e.g. azathioprine) or dicumarol administration

TABLE VI
INDICATIONS FOR ALLOPURINOL

1. Tophaceous gout
2. Gout with renal insufficiency (Creatinine clearance <60 ml/min)
3. Urate excretion >800 mg/d
4. History of renal calculi
5. Secondary hyperuricemia with overproduction of uric acid
6. Allergy to uricosurics
7. Uricosurics ineffective or poorly tolerated

approximately 6.8 mg/dl. Identification of patients who overproduce or underexcrete uric acid will guide appropriate drug therapy. Patients with hyperuricemia excreting 600 mg or less of urinary uric acid/day are classified as underexcreters and those excreting more than 800 mg per day are considered overproducers. These urine collections must be done in the absence of antihyperuricemic drugs and on a normal to low purine diet (organ meats such as liver or pancreas are high in purine content).

Simkin (1977) has reported that equivalent information may be quite readily obtained by collecting a single morning untimed urine specimen and measuring urinary uric acid and creatinine as well as a serum creatinine. One then multiplies the urinary uric acid by the serum creatinine and divides this number by the urinary creatinine (all in mg/dl); a value greater than 0.7 indicates that the patient is an overproducer of uric acid.

$$\frac{U_{ua}\frac{(mg)}{(dl)} \cdot S_{Cr}\frac{(mg)}{(dl)}}{U_{Cr}\frac{(mg)}{(dl)}} > 0.7 = \text{overproducer}$$

For patients with normal renal function who are underexcreters of uric acid, a uricosuric drug such as probenecid is appropriate *(Table V)*. Allopurinol is the drug of choice for overproducers of uric acid and is specifically indicated in patients with renal insufficiency, a history of urate stones, or tophaceous gout; this drug inhibits xanthine oxidase which converts hypoxanthine and xanthine to uric acid. In the presence of renal insufficiency, the dose of allopurinol should be reduced because it is metabolized by the liver and excreted by the kidney. This drug can be given in a single daily dose and most patients are controlled in a range from 300-600 mg/day. The dose of allopurinol should be reduced when it is given concomitantly with dicumarol or with azathioprine or 6-mercaptopurine (the latter two drugs are metabolized by xanthine oxidase). Colchicine should be continued during this initial period (about six weeks) of antihyperuricemic therapy because recurrent attacks of inflammatory arthritis may occur with changing serum uric acid levels. A rare patient may require both a uricosuric agent and allopurinol to lower his serum uric acid below 7 mg/dl.

Diuretic-induced hyperuricemia <9-10 mg/dl should not require antihyperuricemic therapy, as these patients rarely develop gouty arthritis or chronic interstitial nephritis. Above 10 mg/dl most rheumatologists would favor treatment with allopurinol, although there may be a higher incidence of adverse reactions when allopurinol and a thiazide are used together compared with allopurinol alone. Probenecid interferes with the naturetic (diuretic) effect of most common diuretic agents; thus these drugs should not be used concomitantly. Shortly to be available is a new diuretic drug, tricrynafen, which remains uricosuric even with long-term administration.

Hypouricemia *(Table III)* occurs infrequently, and is most commonly due to high dose salicylates (>2 gm/d). Paradoxically, low dose salicylates (<2 gm/d) raise the serum uric acid by inhibiting its renal tubular secretion. Other agents known to be uricosuric include radio-opaque dyes, guaifenesin (Robitussin®), dicumarol and phenylbutazone. Patients with lung tumors or lymphomas may present with hypouricemia. Dietary restrictions are relatively ineffective in treating hyperuricemia. Although patients with gout should avoid eating high purine foods — such as liver or pancreas — and consuming alcoholic beverages in excess (lactic acid inhibits renal tubular secretion of urate), an unpalatable low purine diet rarely lowers the serum uric acid by more than one mg/dl.

Although the treatment of asymptomatic hyperuricemia remains controversial, drug therapy

for patients with primary hyperuricemia is here recommended only for patients whose serum uric acid exceeds 9 mg/dl, or with serum uric acids of >7 mg/dl associated with high urinary uric acid excretion (>800 mg/d). This latter situation predisposes to formation of renal stones. In hyperuricemia secondary to specific diseases such as hematopoietic disorders, psoriasis or chronic renal disease, patients should receive antihyperuricemic therapy only if they are overproducers of uric acid. In hematologic malignancies allopurinol should be administered prior to initiation of cytotoxic therapy to prevent massive deposition of uric acid in renal tubules.

Treatment initiated for primary hyperuricemia is essentially lifelong. The physician should see these patients at least twice yearly to monitor the serum uric acid and to remind them to take their medication regularly. Fluid intake of at least 2 liters/day is also desirable in order to minimize urate stone formation. Patient education is an essential part of a successful therapeutic program for these asymptomatic individuals.

In summary, the essential points of management of gout and hyperuricemia include: (1) accurate diagnosis, preferably by demonstration of uric acid crystals in synovial fluid or tophi; (2) anti-inflammatory drugs for an acute gouty attack; (3) prophylactic colchicine to prevent recurrence of gouty arthritis; and (4) antihyperuricemic drugs to maintain the serum uric acid below 7 mg/dl. The goal — not yet achieved — is to prevent or retard joint destruction by acute inflammatory and chronic tophaceous gout, and to minimize the development of chronic interstitial nephritis secondary to urate deposition within the renal interstitium.

Self-Assessment Questions

True or False

1. The most common cause of hypouricemia is administration of high dose salicylates.
2. Allopurinol is a potent anti-inflammatory agent used in the treatment of acute gout.
3. A single mid-morning urinary uric acid "clearance" will identify those patients who overproduce uric acid.
4. The presence of negatively birefringent intracellular synovial fluid crystals permits one to make a definitive diagnosis of gout.
5. In the drug treatment of hyperuricemia, allopurinol is the first drug of choice.

(Answers on page 624)

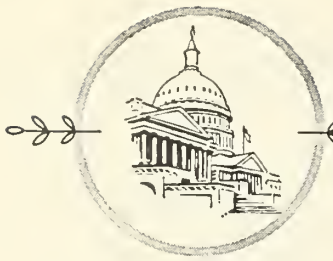
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Socio-ECONOMICS

Disadvantages of Incorporating

While all previous articles written for this column have discussed the many advantages of incorporating a professional practice, it is equally important that the professional fully understand the problems that may be created by incorporation.

Excess Compensation

Upon incorporation, a new tax entity is created that has a separate legal existence. One of the advantages in creating this entity is the fact that monies can be accumulated at a lower tax rate than individually. This presents the dilemma of how to recover this money from the corporation without it being declared "excess compensation" by the Internal Revenue Service (IRS). If in any tax year an individual's income from the corporation should exceed billings, the IRS may challenge such amounts as "excess compensation." If this happens, the corporation loses the deduction for any amount paid out, and the individual becomes liable for personal taxes on that income. This amount will be treated as unearned income and may be taxed at a 70 per cent rate.

Benefit Plan Administration

While the most attractive tax advantage to incorporating may be found in the many fringe benefits the corporation can provide, an understanding of the possible hazards is most important. The IRS provides that fringe benefit expenses may be deducted by the corporation only if they are provided on a non-discriminatory basis to all full-time employees. They define "full-time" as any person who has completed 1000 hours of service in the preceding tax year. It thus becomes possible to include "part-time employees" in employee benefit plans. It is also necessary to note that these benefit plans must be provided on a non-discriminatory basis. This is generally interpreted to mean "in relation to income." As an example, assume a physician with a $50,000 salary from his corporation purchases a group life insurance policy for himself in the amount of $50,000, or 100 per cent of his salary. To qualify this expense for a corporate deduction, an equal benefit (100% of salary) must be provided to *all* full-time employees. Obviously, a professional with three or four long term employees could quickly lose any tax advantages because of the additional expense for his employees.

Operating Expenses

While incorporating a practice could provide significant tax savings, there are instances in which the expenses of operating the corporation could negate any savings. While these instances are rare, they are possible. Generally legal, accounting, consulting, insurance, and the miscellaneous expenses of operating the corporation will average about $1,500/year.

Corporate Divorce

While the problems facing the break-up of the professional corporation are essentially the same as those facing any partnership, they can become extremely complex and expensive for the corporation and its stockholders. If the departing stockholder is hostile, many legal and tax problems can be created for the remaining stockholders, possibly even forcing a corporate liquidation. Generally the problems of "corporate divorce" can be avoided by following two basic rules: (1) do not incorporate a partnership until it has been operating for at least three years; and (2) do not bring any new stockholders into the corporation until they have been employed by the corporation for at least three years. This three-year

(Continued on page 624)

Further information may be obtained from Midwest Pension Planners, Mr. John E. Lyons, Plan Consultant, 1010 Merchants National Bank Bldg., Topeka, KS 66612.

Involuntary Practice Closing

Ed Note: This is the seventh in the series of articles prepared by the AMA Department of Practice Management, Division of Medical Practice. For other articles on this topic, see the following issues: November and December 1977, and January, February, March, July, and September 1978.

The subject of practice management has become more critical in recent years. To assist the physician in a smoother operation of his office, the Department has produced workshops for medical assistants dealing with such aspects as collections, public relations-telephone techniques and office management, as well as practice management workshops for the new physicians. The Kansas Medical Society Executive Office will present such workshops upon demand by members.

"How to Start to Stop" (Sept. '78) dwelt on aspects involved in "winding down" a medical practice with retirement as the ultimate goal. But what happens when the physician dies — suddenly, through illness or an accident?

Generally the spouse has to assume the brunt of dismantling and closing the medical practice. Many of the tasks have to be done when the spouse is under great emotional strain and there is a lack of time for making sound decisions.

However, advance planning can alleviate this situation. Planning for an emergency that may never happen is no more gruesome than purchasing life or accident insurance. And there is no cost for most of the things you can do that can be of real help to your spouse if a tragic situation does occur. Planning in advance will give you assurance that matters will be attended to in the way you prefer.

The first and easiest — but most important — is to prepare a list of "know abouts." These are the simple things that the surviving spouse should know. (You will find a list with this article.) Some are so obvious as to not seem worth writing down — but many physicians have not provided this simple help for their spouses.

For example, recently when a doctor died no one could find the will. The wife knew that her husband had prepared a will; the lawyer remembered writing one, but the document itself couldn't be found. The attorney thought the doctor had filed it at his office. The medical assistants at the office thought the doctor had taken it home, and the wife thought her husband had left it in the possession of the attorney. Eventually it was found, but for about ten days the wife, with all of her other problems, was faced with the uncertainty of how the estate would be settled.

In another instance the doctor had a small strongbox in his office-den at home. His wife knew about it and also that he kept insurance policies and similar valuable papers in it. What she didn't know was the combination. Not that this was any secret; he had simply never thought to tell her where it was written down. It was a steel box, about a foot and a half square, with double walls about an inch thick for fire protection. When the doctor died suddenly, his wife knew where to look for some of the papers she needed — but didn't know how to get into the box. One of her sons wanted to take a sledge hammer and knock the combination off and then try to pry it open with a crowbar. Another suggestion was to have a welder cut it open with an acetylene torch. A professional locksmith finally got the box open, but it was an experience the widow could have done without.

Of course your spouse knows where you keep the key to your safety deposit box — or does she? We know of an instance where the wife of the physician knew he had a lockbox. He kept a few things in it for her along with other valuable papers. He just forgot to tell her where he kept the key. At his death, arrangements finally had to be made with the bank to drill it open.

Any advance planning assumes an up-to-date will. This is a must. It should be reviewed every few years to provide for changes in your family, your financial situation, and for any geographical changes you might have made — moving from one state to another.

It is not macabre for a husband and wife to talk about funeral arrangements. It is probably not a subject that you would care to dwell on, but the purchase of a cemetery plot, simply as a business transaction, is much easier and better than for a surviving spouse to have to secure one in an emergency situation. If one or the other has some very definite ideas about burial, cremation or providing body parts for transplant or medical research, the wishes of each should be written down and filed with the will. (Four items are listed under the attachment headed Sudden Death.)

Some things should be done quickly in case of the sudden death of a physician. Collection action on outstanding accounts receivable should be started at once. Any delay in collecting these accounts will radically reduce their value. Arrangements have to be made to pay the office bills, to check on taxes owed, to discontinue services of employees, and to find out about the office lease or rental contract. Patients have to be notified, arrangements made for disposition of medical records, and many of the other

Department of Practice Management, Division of Medical Practice, American Medical Association.

matters that are included in the orderly preparation of a voluntary practice closing.

A medical practice may of course be: (1) abandoned; (2) given away; or (3) sold. There is nothing wrong with any of the three approaches, but the third choice would seem preferable. This assumes a solo practice rather than an ongoing partnership or corporation. With either a partnership or multi-doctor corporation arrangements for both voluntary and involuntary withdrawal from practice is (or should be) a part of the partnership or corporation regulations.

Selling a practice outright is much more difficult. A future article will be devoted to this subject.

KNOW ABOUT

1. Know location of last executed will, trust agreements, codicils, etc.
2. Know location of lockbox keys and any safe combinations.
3. Know location of personal and business bank accounts, savings accounts and checking account bank ID numbers.
4. Know location of birth certificates, marriage certificate, citizenship papers (if applicable), military discharge papers, adoption papers, Veterans Administration claim number, and Social Security number.
5. Know name, address and telephone number of attorney, medical management consultant, accountant, insurance agent, stock broker, and real estate agent with whom business has been transacted.
6. Know location of titles and mortgage of home and any other applicable real estate contracts, leases, or rental agreements.
7. Know location of all insurance policies and numbers.
8. Know location of stock and bond securities with cost information, including pension program information.
9. List credit card names, numbers and addresses.
10. List associations, specialty societies and professional, fraternal, and social organizations in which membership is maintained.
11. Location of unpaid personal bills and other outstanding obligations.

SUDDEN DEATH

1. Notify office manager or receptionist and answering service with information to provide callers.
2. Notify attorney, medical management consultant, or accountant who has been serving the practice.
3. Notify funeral director and clergy.
4. Have the location of the cemetery lot, plot number, and deed available when talking with the funeral director. In case of cremation, the disposition of ashes.

Physician Distribution

(Continued from page 594)

ceived windfall repayment of their education loans by the Federal Government since they would have established their practices in those shortage areas anyway.''

The report continued:

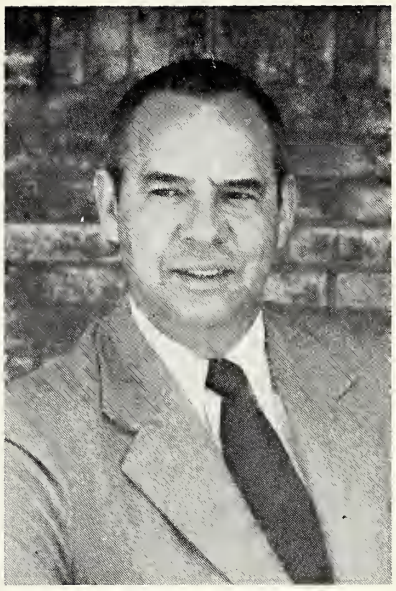
''The Congress should also reconsider the necessity for HEW to complete its study on physician extender reimbursement as required by the Social Security Amendments of 1972 in view of the ''recent legislation enacted that provides for: (1) Medicare (part B) and Medicaid reimbursement for physician extender (physician assistants and nurse practitioners) services rendered in certain rural health clinics in medically underserved areas; and (2) demonstration projects to be conducted with respect to reimbursement for services provided by physician-directed clinics which employ physician assistants and nurse practitioners.''

HEW, which agreed with most of the GAO recommendations, said it was also concerned about the possible underuse of Corps physicians and that it must consider alternative health care delivery modes or staffing arrangements for those HEW-designated shortage areas which cannot sustain full-time medical practices or retain physicians. HEW expects some progress toward improved use to occur as major barriers to the use of physician extenders are reduced (*i.e.*, eligibility for reimbursement and limitations of licensure).

HEW also stated that existing National Health Service Corps sites will not be continued if sufficient support for the Corps project has not been demonstrated over the period of Corps involvement.

The GAO said it had considered comments made by officials of the programs and projects reviewed, and those of the American Medical Association, in preparing its report.

The President's Message



Negotiations

Jerry Slaughter and I attended an AMA seminar on "The Physician Negotiator, The Dynamics of Conflict Resolution," at Lake Arrowhead, California, September 21-23. It was a most rewarding experience and we learned much about the art of negotiation and practiced our new skills in mock negotiation sessions. The most important thing we learned was that most doctors are nice guys, play by the Marquis of Queensbury rules, and almost always lose! BUT IT DOESN'T HAVE TO BE THAT WAY! The art of negotiation is probably the most important new skill we can learn, and should acquire as soon as possible.

There is no doubt that these skills will be called into play in the near future. It has already begun between housestaffs and hospital administrations; in fact, several residents, and officers in the AMA Student Business Section, were in attendance at the seminar.

Hospital based physicians are also increasingly coming to the bargaining or negotiating table with hospitals and third parties over a variety of issues that are due to become more complex. We had a good number of pathologists, radiologists, and anesthesiologists at the seminar. Paige Clousson, Director of the AMA Department of Negotiations, spoke of attending similar seminars sponsored by the American Hospital Association and hearing hospital administrators advised to keep control of their medical staffs by the "divide and conquer" principle through negotiations with the hospital based physicians.

Plastic surgeons were also much in evidence at the seminar. The recent Federal Trade Commission action against their Society for restraint of trade has made them feel the need to become proficient in this area.

The September 18th issue of *Medical Economics* carried a story of HEW's Health Care Financing Administration already at work, behind the scenes, "—Developing a plan that would wipe out 'usual, customary and reasonable' fee reimbursement under Medicare and Medicaid and replace the UCR system with a series of fee schedules NEGOTIATED among doctors and carriers. — Ultimately such a fee schedule would undoubtedly regulate all doctors' fees under National Health Insurance. — Practicing physicians would be appointed to negotiating groups that would hammer out fee schedules with representatives of carriers and perhaps of 'the public — prospective patients.'" Although Mr. Robert Derzon has been replaced, Secretary Califano remains and the plan is his.

Senator Kennedy's proposal for National Health Insurance also includes fee schedules NEGOTIATED among state or county medical societies, HMOs, IPAs, or other groups of doctors and the federal government. There can be little doubt left that negotiations will play an increasingly important role in our professional lives in the near future.

At the seminar, Jerry and I "negotiated" an agreement with Jerry Clousson to conduct the same seminar in Kansas if we could enroll at least 25 doctors for the course. I personally believe we can have a good deal more than that number. I urge you to give serious consideration to attending and learning the art of negotiation when you receive the notice for this session. It will help in many areas in addition to your professional activities and responsibilities.

Fraternally yours,

Warren E. Mayer, M.D.

President



Editorial COMMENT

The Unimpaired Physician

This issue of the JOURNAL focuses on a phenomenon of considerable significance, the Kansas Medical Society's Program for the Impaired Physician. The significance lies in the fact that this program, along with similar ones being developed throughout the country and fostered nationally by the AMA, represents, to the best of our knowledge, the first venture of a professional entity in establishing an organized effort to identify and salvage those members whose professional capabilities have been damaged by physical, mental, or behavioral defects.

There have always been efforts, of course, to control the competence of medical practitioners — beginning with the physician's conscience — and there has been official recognition of physician fallibility at least since the days of Hammurabi. Those efforts which have developed within the group have been concerned with the quality of the service. Those which have developed outside have primarily implemented the social and legal maneuvers of accepting the physician into service, and have embodied the restrictive or punitive measures presumed to maintain that service at an acceptable level. But between these limits of acceptance and rejection, there has been little firm or consistent attention to the physician's individual health problems and their impact on his practice.

So organizational recognition that "impaired" physicians should be identified and that the assistance of their colleagues in meeting their problems should be a formally sponsored function of the organization establishes a new level of maturity in the expression of professional responsibility. The term, "impaired," has received, rather quickly, the blessing of acceptance as an appropriate designation for the subjects of the program. One reason for this is that it is comfortably inclusive. It can be interpreted to refer, depending upon the user's wish, to those physicians who are addicted to alcohol or drugs (who have been the primary focus of the effort) but it can also apply to those who, by age, trauma, illness or similar disabilities, are unable to maintain a previous level of function and must be removed, either to another level of service or from the scene, for the welfare of their patients, themselves, and the profession. So the inclusive nature of the term thus endows it with a euphemism which makes it easier to refer to the physician-addict without implying prejudice or accusation. There can be little doubt, however, as to the group of primary interest if we note how consistently the term is limited to the physician-addict group and we must acknowledge the value of the term in easing our communication. The slow pace with which the profession has come to its current action seems to have been a counterpart of the denial phase characteristic of these afflicted individuals — even though we were rather consistently confronted with the evidence, it has taken us a while to admit that physicians could be drunks and dope addicts. Perhaps we have been waiting for just such a term.

This physically disabled group (a convenient though not entirely satisfactory designation) shares with the addicted members the common denominator of disability, but the numerators are different and varied. The profession has always felt more comfortable with the strictly physical disabilities which are more apt to excite compassion than censure. The physically disabled physician often represents that highest achievement of medical service, the coronary or stroke victim, that paragon of virtuous overwork which the profession admires. Fundamentally, the relationship of this individual with his colleagues is different from that of the physician-addict. He asks for assistance because he has no choice and his disability is obvious. His personal role in regard to his affliction is primarily accommodative. His colleagues participate on a therapeutic and rehabilitative level but this is familiar territory for them and once the objective of stability is attained, the patient can be left to his own care. The condition is self-contained, the efforts to meet it are open and admired, and the ultimate incapacity

can be accepted as a regrettable but understandable condition.

But the physician-addict poses more difficult problems. Since his behavioral deviations, initially at least, can be blended in with the social norms more readily, he may well go undetected — even by himself — for some time. Recognition and confrontation by family and colleagues meet denial, deprecation, and resistance. The colleagues have their confrontations too — they must face the question not only of their personal roles in the matter but the hazard posed by permitting continued practice by the addict. It is of interest that recovered physician alcoholics and addicts report that their colleagues almost invariably contribute to a delay in effective therapy (unintentionally, to be sure) by their insistance upon acting as if the afflicted physician was a different breed from lay patients, and either not pursuing or using differently those medical measures they would apply immediately to their non-physician patients.

This brings us to an aspect of the program which is obscured by the attention to the principals, the fact that only slightly below the impaired physician on the list of beneficiaries is the unimpaired physician, this colleague who is a member of the formless and largely ineffectual group which has tried to cope with the external aspects of the problem — usually on a friendship basis but almost certainly an example of too little and too late. The structure of medical practice, the availability of addictive agents, and the areas of independent function present in any physician's life enhance the opportunities for undetected alcohol or drug indulgence. Even when some misuse is apparent, the episodic and clandestine nature of its occurrence, along with the combined action of wishful thinking and desire to avoid involvement, can be sufficient to divert even the concerned colleague from confronting the matter realistically — not to mention an ignorance of how to proceed.

There may be times of entreaty and argument, antagonistic reaction or remorseful protestations of improvement, but the general pattern has been one of gradual deterioration of the individual until the first real interaction was one of a disciplinary nature. Any physician who has been involved in such sessions knows the sense of sadness and frustration that the relatively naive colleague feels in trying to meet the denials, evasions, anger, and promises. Such sessions, however phrased, are perceived by the bedeviled subject to be threats (which they usually are). Once the hearing is over, the group (except for that individual) goes quickly back to its less agonizing endeavors with the hope that he will solve the problem, possibly by achieving the promised recovery or at least removing himself from its jurisdiction, but with the unspoken admission that the group effort was probably of little avail and the next encounter will be unreservedly punitive.

Now it would be in the same order of evasive and wishful thinking to presume that the establishment of this altruistically practical program was going to bring some miracle cure to the situation. The social and professional stresses are going to continue to assault physicians and some will resort to pharmacologic defenses while others rely on scarcely less destructive but more acceptable mechanisms. The establishment of this program means that for the impaired physician a path is opened to a concerned, nonjudgmental, professionally knowledgeable area of assistance, and it is hoped that the awareness of this availability will result in earlier acceptance of its services. But at the same time, it significantly reinforces the position of the colleague who has too often been sitting by as a sorrowful, angry, and frustrated witness to a tragic human loss, impotent to meet the situation with anything other than his anxiety and desire to help.

The success of the plan, then, will be measured in one way — the effective reaching of the impaired physician early — even before there are overt indications of the impairment. By establishing, as a backdrop for the committee's function, a sincere and informed atmosphere of interest in helping such individuals, the plan will encourage a certain number of involved physicians to avail themselves of this assistance. It is by conscious intent that recovered alcoholics and addicts will be incorporated as fully as possible in the program, for it has been amply demonstrated that no one can reach these people as effectively as those who have been reached. But the unimpaired physician's early detection of the physician-addict and an informed support during the rehabilitative phase will require a comparable dedication on his part — an awareness of the impaired physician's problem but an equal acceptance of his own problem in knowing his feelings about his impaired colleague. He will now have the benefit of an organizational structure and method with trained and experienced guidance when he is confronted with the problem. It may well be that the response of the unimpaired physician will be as crucial a factor in the program's success as that of the subject.—*D.E.G.*

Eminently Treatable Disorders

(Continued from page 617)

Answers

1. *True.*
2. *False.*
3. *True.*
4. *True.* However, prior injection of intra-articular crystalline glucocorticoids may mimic urate crystals.
5. *False.* For an underexcreter of uric acid with normal renal function probenecid is an older and safer drug to use. If a patient is unresponsive to, or intolerant of, the drug, then allopurinol is indicated.

Disadvantages of Incorporating

(Continued from page 618)

incubation period will allow sufficient time to anticipate most problems.

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In summary, incorporating a practice may generate significant tax savings today, but it may also generate significant headaches tomorrow. It is essential to evaluate the legal and tax consequences five or ten years into the future. Usually, the advantages far outweigh the disadvantages, but incorporating a practice requires careful evaluation and planning.

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CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).
WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect, rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence:* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy:* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children:* Tenuate is not recommended for use in children under 12 years of age.
PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.
ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema. *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.
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References: 1. Citations available on request—Medical Research Department, MERRELL RESEARCH CENTER, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215 2. Hoekenga, M.T., O'Dillon, R.H., and Leyland, H.M.: A Comprehensive Review of Diethylpropion Hydrochloride. International Symposium on Central Mechanisms of Anorectic Drugs, Florence, Italy, Jan. 20-21, 1977.

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Alcoholism, other drug abuse, and medical/neurological/ psychological problems are potentially treatable conditions. All impaired physicians should be encouraged to seek help at the earliest possible time in order to retain or regain full effectiveness to practice medicine. Please contact one of the following:

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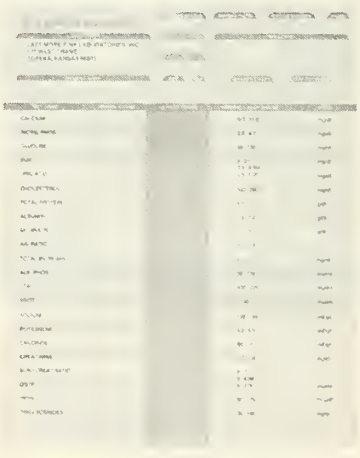
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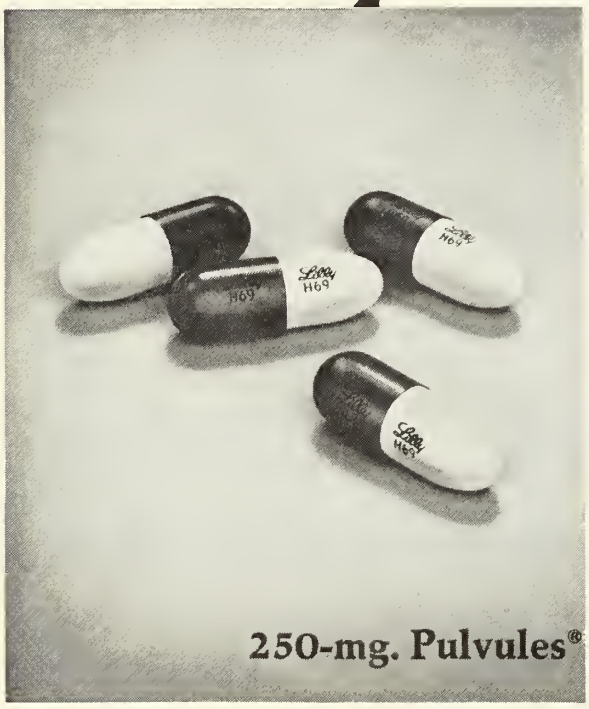
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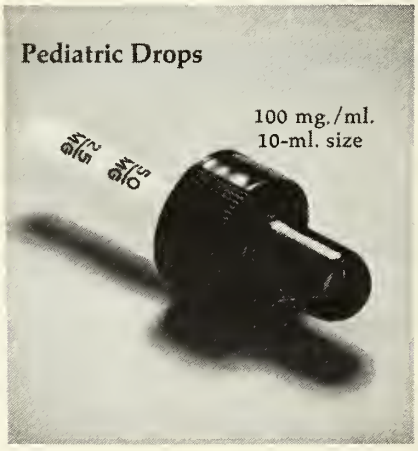
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Warnings: As with most CNS-acting drugs, caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Withdrawal symptoms (similar to those with barbiturates, alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal/muscle cramps, vomiting, sweating). Keep addiction-prone individuals (drug addicts or alcoholics) under careful surveillance because of predisposition to habituation/dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations, as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

ORAL: Advise patients against simultaneous ingestion of alcohol and other CNS depressants.

Not of value in treatment of psychotic patients; should not be employed in lieu of appropriate treatment. When using oral form adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increase in dosage of standard anticonvulsant medication; abrupt withdrawal in such cases may be associated with temporary increase in frequency and/or severity of seizures.

INJECTABLE: *To reduce the possibility of venous thrombosis, phlebitis, local irritation, swelling, and, rarely, vascular impairment when used I.V.: inject slowly, taking at least one minute for each 5 mg (1 ml) given; do not use small veins, i.e., dorsum of hand or wrist; use extreme care to avoid intra-arterial administration or extravasation. Do not mix or dilute Valium with other solutions or drugs in syringe or infusion flask. If it is not feasible to administer Valium directly I.V., it may be injected slowly through the infusion tubing as close as possible to the vein insertion.*

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Withdrawal symptoms (similar to those with barbiturates, alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal/muscle cramps, vomiting, sweating). Keep addiction-prone individuals under careful surveillance because of predisposition to habituation/dependence. Not recommended for OB use.

Efficacy/safety not established in necnates (age 30 days or less); prolonged CNS depression observed. In children, give slowly (up to 0.25 mg/kg over 3 minutes) to avoid apnea or prolonged somnolence; can be repeated after 15 to 30 minutes. If no relief after third administration, appropriate adjunctive therapy is recommended.

Precautions: If combined with other psychotropics or anticonvulsants, carefully consider individual pharmacologic effects—particularly with known compounds which may potentiate action of Valium (diazepam), *i.e.*, phenothiazines, narcotics, barbiturates, MAO inhibitors and antidepressants. Protective measures indicated in highly anxious patients with accompanying depression who may have suicidal tendencies. Observe usual precautions in impaired hepatic function; avoid accumulation in patients with compromised kidney function. Limit oral dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation (initially 2 to 2½ mg once or twice daily, increasing gradually as needed or tolerated).

INJECTABLE. Although promptly controlled, seizures may return; readminister if necessary; not recommended for long-term maintenance therapy. Laryngospasm/increased cough reflex are possible during peroral endoscopic procedures; use topical anesthetic, have necessary countermeasures available. Hypotension or muscular weakness possible, particularly when used with narcotics, barbiturates or alcohol. Use lower doses (2 to 5 mg) for elderly/debilitated.

Adverse Reactions: Side effects most commonly reported were drowsiness, fatigue, ataxia. Infrequently encountered were confusion, constipation, depression, diplopia, dysarthria, headache, hypotension, incontinence, jaundice, changes in libido, nausea, changes in salivation, skin rash, slurred speech, tremor, urinary retention, vertigo, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances and stimulation have been reported; should these occur, discontinue drug.

Because of isolated reports of neutropenia and jaundice, periodic blood counts, liver function tests advisable during long-term therapy. Minor changes in EEG patterns, usually low-voltage fast activity, have been observed in patients during and after Valium (diazepam) therapy and are of no known significance.

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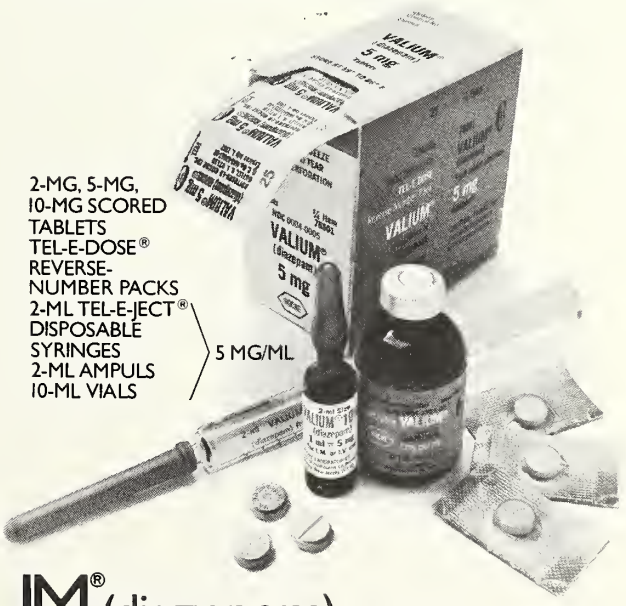
In peroral endoscopic procedures, coughing, depressed respiration, dyspnea, hyperventilation, laryngospasm/pain in throat or chest have been reported.

Management of Overdosage: Manifestations include somnolence, confusion, coma, diminished reflexes. Monitor respiration, pulse, blood pressure; employ general supportive measures, I.V. fluids, adequate airway. Use levarterenol or metaraminol for hypotension, caffeine and sodium benzoate for CNS-depressive effects. Dialysis is of limited value.

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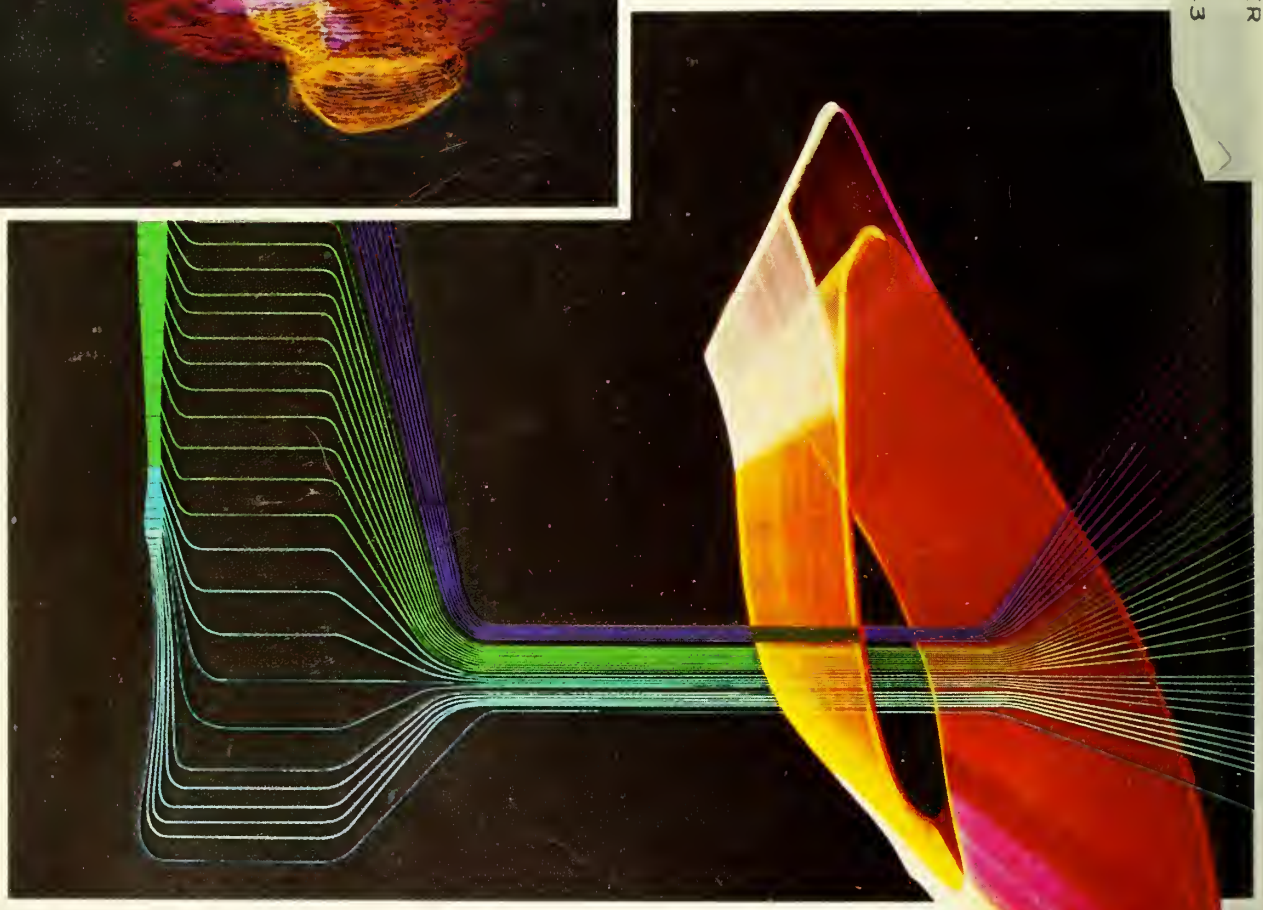
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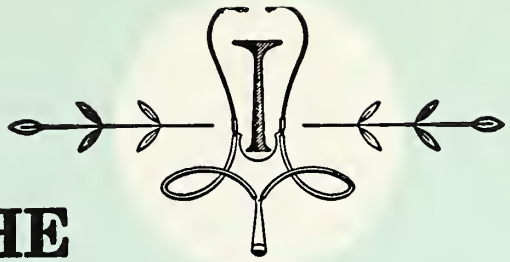
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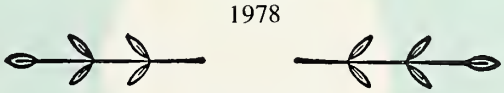
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The JOURNAL of the KANSAS MEDICAL SOCIETY

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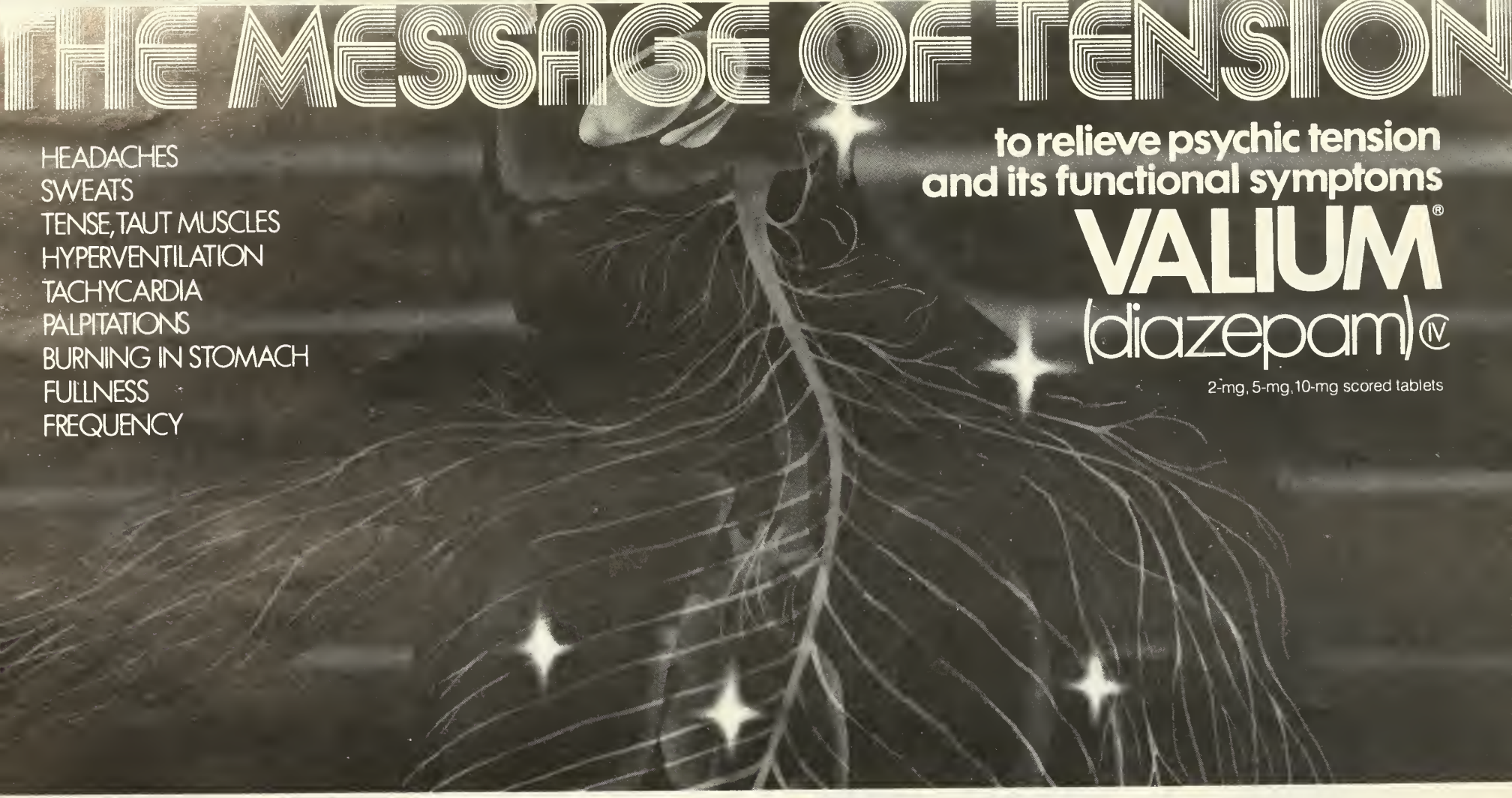
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Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed, drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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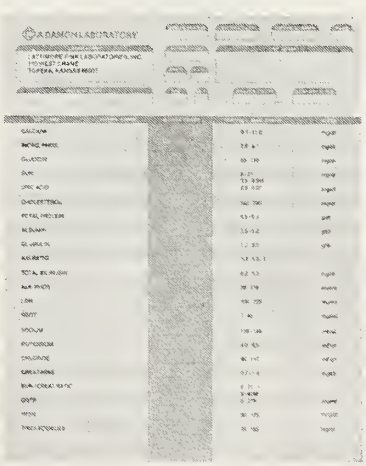
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Adverse Reactions: On rare occasions oral administration of the drug has been associated in time with the occurrence of hypotension, tachycardia, nausea, vomiting, dizziness, abdominal distress, and severe rash. If rash appears the drug should be discontinued.

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CME

Those Other Categories: An Arithmetic Lesson

(This article is addressed to all physicians who mutter and curse their state licensing board or medical society for establishing CME requirements. It can be used as a handout when you hear cries of despair about not having time to meet CME mandates. Reprinted from *The Journal of the Medical Association of the State of Alabama,* February 1978.)

WHAT'S the first impression that comes into your mind when someone says, "CME?" Do you envision a large gathering of physicians seated quietly in neat rows of chairs, taking notes and carefully listening as an august professor from a nearby medical school presents a formal lecture — liberally illustrated with colored 35 mm slides? Is this close to your impression? From talking to many physicians, I get the feeling that it is. CME means Category 1 "live" programs — right? Certainly this kind of education is an important part of your personal CME program; AMA requirements for the Physician's Recognition Award (PRA) stipulate that 60 of the 150 hours of CME over a three-year period must be Category 1 (40%). But how about the other 60 per cent?

The rest of this article is a commercial for "those other categories" (2, 3, 4 and 5) which can be very effectively used in meeting 60 per cent of your CME requirements. Attaining CME credit in these categories is both easy and difficult.

First, let's talk about the easy part. Many of you are involved in Category 2-5 CME activities and may not even realize it! To illustrate this, let us go quickly through these categories and do a little arithmetic along the way:

Category 2: CME activities of hospitals, medical societies, etc., which are not formally accredited for Category 1 CME (maximum allowable — 45 hours).

Do you attend a hospital staff meeting once a month where some sort of scientific program is presented? If you do, take credit for 36 hours in Category 2 for the three-year period.

Do you occasionally have scientific programs at your county medical society meetings or at your annual state specialty society gatherings? If you only attend three of these per year, take 9 hours credit for Category 2.

See how relatively painless this is? You probably have been doing these kinds of activities for years, but may not have related them to your CME requirements. Add these examples together for a total of 45 hours — almost one-third of your total CME requirements.

Category 3: Medical Teaching (maximum of 45 hours). You might think that this category only relates to medical school professors or learned visiting lecturers. Not so. Do you ever teach medical students or residents at your hospital? Have you ever presented a program for fellow physicians? How about training sessions you put on for nurses, medics, etc.? All these count for Category 3. I'll bet most of you do at least 5-10 hours over a three-year period.

Category 4: Papers, publications, books and exhibits (45 hours maximum). Let's skip this one since this kind of activity, though very beneficial, may not be as applicable to the majority of physicians.

Category 5: Non-Supervised Individual CME Activities (maximum of 45 hours).

Do you spend at least one hour per month reading scientific journals, books, etc.? If so, take credit for up to 22 hours of Category 5 CME.

Do you spend time in meetings, or programs, concerned with the review and evaluation of patient care — such as peer review, medical and chart audits, case conferences, PSRO meetings, tissue review, infections or death conferences? Take a maximum credit of 22 more hours of Category 5 CME for these activities.

Other hours for taking self-assessment exams and time spent learning from a consultant can also be credited here for Category 5.

Well, that's it — just crediting some of the activities which I feel would be usual for most physicians, will easily give you over a total of 90 hours — the other 60 per cent needed to meet your CME requirements.

Now let's consider the difficult part. Keeping track of activities in these "other categories" can be difficult because you will have to do this yourself.

(Continued on page 657)

George D. Oetting, Ed.D., Director of Education, The Medical Association of the State of Alabama.

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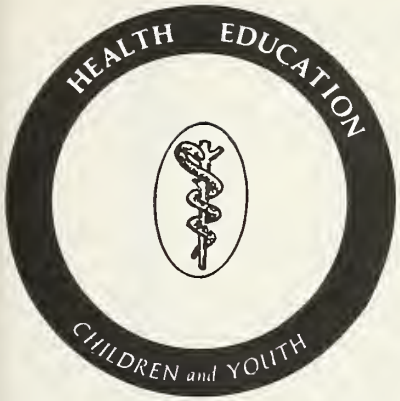
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During September, October and November, I have visited ten county auxiliaries, attended the National Fall Confluence in Chicago, and visited the Missouri state fall board meeting. In addition, we have conducted two workshops attended by representatives from several counties, have had an Executive Board meeting, a Long Range Planning Committee meeting, and a meeting of the Impaired Physicians Committee. At all times, I have been consistently impressed with the dedication and caring of Auxiliary members and their physician spouses.

Our health education programs have been enthusiastically received by all members, and our goal is to gear these ongoing programs to the needs of each individual community. The questionnaire on marriage enrichment sent to each Auxiliary member has provoked positive comment from members, and more than 500 have been filled out and returned to us.

The Fall Confluence, attended by six Kansas representatives, presented some excellent programs in the area of long and short range planning, parliamentary procedure, priority legislation, public relations, alcohol and the teenager, developing healthful lifestyles, health planning and the hospice concept. In January we will have our own Kansas Confluence. This will be a time to share ideas, hear criticisms and suggestions, and bring the sessions from the AMAA Confluence to our Kansas members.

As the first half of a busy year draws to a close, may I take this opportunity to wish all of you the Joy and Blessings of the Christmas Season.

Sincerely,
Jean Crouch
President
Kansas Medical Society Auxiliary

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Council Meeting

Report of Meeting Held October 8, 1978

A meeting of the Council was held on Sunday, October 8, 1978, beginning at 10:00 AM, at the Emporia Holiday Inn. Warren Earl Meyer, M.D., President, called the meeting to order. Present were Drs. Lewis G. Allen, Donald E. Beahm, John Blank, Kenneth Boese, Richard Brownrigg, Clair C. Conard, Jack R. Cooper, Herbert Fransen, Donald Goering, William Halling, Robert P. Hudson, John D. Huff, Robert Hughes, Warren E. Meyer, Herbert M. Nason, Alex Scott, Marvin Snowbarger, M. C. Spencer, W. K. Walker, Roger Warren, Kermit Wedel, Emerson Yoder.

Also present were Jerry Slaughter, Gary Caruthers, and Val Braun.

Doctor Meyer thanked the members for attending. He reported that Mr. Wayne Stratton, KMS Legal Counsel, advises that the U. S. Supreme Court had refused to hear the appeal from Dr. Liggett concerning the constitutional test of the Health Care Stabilization Fund law. Therefore, it appears that the law is constitutional.

Val Braun reported on the KMS Impaired Physicians Program. She briefly reviewed the history and formation of the committee, including legislation that has been enacted this year. The committee has open communications with the Kansas State Board of Healing Arts. KMS Auxiliary is actively involved in committee efforts. The emphasis of the program in 1979 will be on early identification and referral of impaired physicians, with a concerted educational effort directed as MDs, hospital medical staffs, Auxiliary, paramedical groups, and lay public. The committee has proposed a 1979 budget of $20,000. This would primarily consist of expenditures for contact/support team work, a training workshop, meeting expenses, telephone/supplies, staff time, etc.

The Council members reviewed and approved the Executive Committee actions for period March-August 1978.

Jerry Slaughter reviewed the criteria for AMA dues billing and remittance. Because the KMS Executive Office is in the process of implementing a computer billing system, it may be difficult to comply with all of the AMA criteria this year. The Council approved the KMS criteria for billing prior to attempting the AMA system. A request will be made of the AMA to waive compliance in Kansas for one year.

Doctor Walker, Treasurer, reviewed the proposed 1979 budget, specifically referring to the anticipated increases in legal fees, the contribution to Mediserve, Inc., salary increases, and the proposed increase for the activities of the Impaired Physicians Program. Dr. Walker stated that Kansas dues rank fourth lowest of all states, and that they had remained at $125 since 1974.

Doctor Meyer's recommended salary increases for the executive staff were approved. The Council also authorized the President and the Executive Director to determine the salary levels of the secretarial staff. All salary increases will be effective January 1, 1979.

Action was taken to increase dues by $25, to $150, for 1979, and to approve the budget as presented.

Doctor Meyer reported that several requests had been received from committee chairmen to be reimbursed for their expenses in connection with committee work. There is no official KMS policy concerning this matter and traditionally such expenses have not been reimbursed. The Council authorized the Executive Committee to study the question of committee chairmen expenses, and to present a recommendation at a later date.

The AMA request for review of HR-1818, its National Health Insurance bill, was discussed. Doctor Conard, AMA Delegate, explained that neither the AMA nor the Kansas Medical Society favor national health insurance, but that political considerations necessitate the introduction of an alternative proposal. With a minor change, the statement developed by Kansas AMA delegates and alternates was approved.

The Council's attention was called to a news article which had erroneously indicated that the Kansas Medical Society supported state regulation of hospital rates. A letter to the editor clarifying the KMS position on the issue was read to the Council.

Prospective rate review is consistent with the views of the Kansas Hospital Association in utilizing the existing system of Blue Cross-Blue Shield. This question will be discussed at the special Fall meeting of KMS House of Delegates.

★

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Gary Caruthers reported on the activities of the Insurance Affairs Committee. The guaranteed issue group life insurance program was unsuccessful because of insufficient interest by physicians. The Workman's Compensation group savings program has realized a savings of 25 per cent on the premiums during its first year of operation. The committee considered a proposal from a consultant firm to manage retirement programs, but decided not to become involved in endorsing management consultant organizations.

A new format for annual meetings was presented. The 1979 session would be held at the Hutchinson Holidome, beginning on Friday, May 3, and concluding on Sunday, May 6. The Sports Day would precede the session on Thursday, May 2, for those who are interested in sports activities. Because of a lack of exhibit space, no commercial exhibits would be presented. To offset this loss of revenue, a $25 registration fee for the scientific sessions, including lunch, was proposed. Also, former commercial exhibitors will be asked to make voluntary contributions. The proposed format was adopted, with further suggestion that a study be made at the 1979 session regarding the commercial exhibits, for final Council consideration at a later date.

Doctor Goering presented the locum tenens report, stating that the AMA Council on Medical Education discussed the Kansas resolution, seemed to favor it, and will probably submit a resolution or a report on this subject to the next AMA House of Delegates meeting. The purpose of the resolution was to allow time spent in locum tenens by residents to count for educational value toward their matriculation.

Doctor Scott, KaMPAC Acting Chairman, reported that Dr. Antonio Huaman resigned as chairman of KaMPAC. The Board will meet in January 1979, at which time a new chairman will be elected.

A complaint filed by Common Cause contends that AMPAC and KaMPAC are affiliated committees and, thus, are limited to a total contribution of $5,000 to any one federal candidate. The complaint was filed on the basis of AMPAC contribution of $5,000 to two candidates for Congress who received KaMPAC contributions of $2,500 each. As a result of the complaint, the Federal Election Commission has subpoenaed the KMS and KaMPAC records relating to operations, including minutes, bylaws, correspondence, etc. Twenty-three other states are involved in the same complaint.

Election reporting laws now require the reporting of in-kind contributions to political action committees. The Executive Committee was directed to study this question to determine the equitable amount of in-kind contribution (such as staff time) received by PAC.

The long-range planning meeting of the Council has tentatively been set for January 14, 1979. Various issues will be considered at that time, including legislative matters, staffing concerns, and others.

Several Kansas newspapers have indicated that AMA and the Kansas Medical Society support political candidates. Dr. Meyer reported that he has written to the editors of the *Topeka Capital,* notifying the papers that neither AMA nor KMS support political candidates. The support rather comes from the political action committees of these organizations.

A possibility of filing a libel suit against the newspapers was presented to the Council. The consensus was that such action would not be appropriate.

A fall meeting of the House of Delegates has been set for November 12, 1978, at the Hilton Inn, Wichita. Dr. Conard, Speaker, suggested that an agenda listing specific items be prepared and distributed to the appropriate members 20 days prior to the meeting.

It was announced that the Physician Discipline Committee, chaired by Frank Bichlmeier, M.D., is in the process of gathering information from other states and will develop a proposal for presentation either at the House of Delegates or the next Council meeting.

After lunch, Doctor Meyer referred to his letter to Senator Dole, concerning the Federal Trade Commission action against the American College of Plastic and Reconstructive Surgeons. Among other things, the letter deplored the FTC demand that terms "plastic surgery" and "plastic surgeons" be available to any physician wishing to use them.

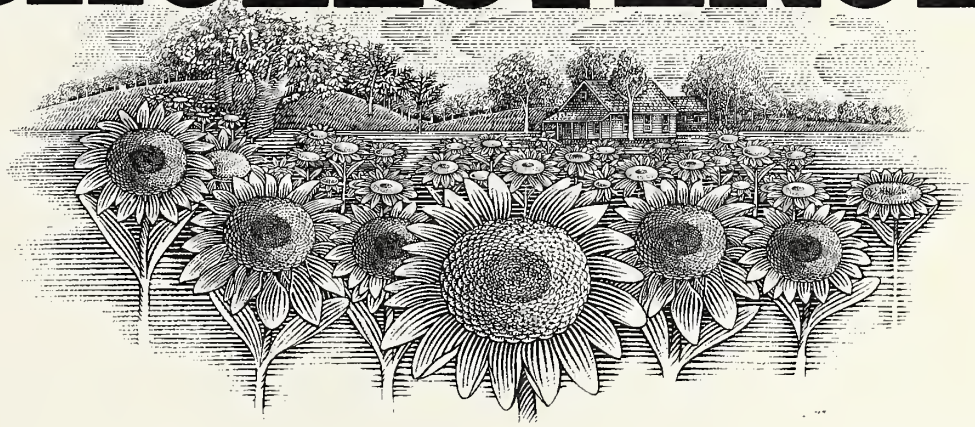
Abuses by Physicians Assistants over the state have been reported. D. Cramer Reed, M.D. has been appointed to chair the committee which would meet with PAs and discuss common problems.

Doctor Meyer recognized Doctor Blank for being honored for 40 years of medical practice in Hutchinson. He congratulated Doctor Blank on behalf of the Kansas Medical Society.

Doctor Nason, President of the Kansas Chapter of the American Academy of Family Physicians, expressed Academy concerns about national health planning guidelines on perinatal care which would affect quality and accessibility of care. Doctor Brownrigg reported that the regulations were changed so that a local HSA could make exceptions if adequately documented.

(Continued on page 648)

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REVISED FORMAT STATE MEETING

IMPORTANT MEETING REMINDER

120th Annual Session of
THE KANSAS MEDICAL SOCIETY

May 3-6, 1979

Holiday Inn-Holidome, Hutchinson

New Format—Thursday-Sunday

Thursday, May 3	Sports Day	
	Sports Banquet	
Friday, May 4	House of Delegates	9:00 a.m.
	Delegates Luncheon	12:30 p.m.
	Reference Committee	2:00 p.m.
	KaMPAC Hospitality Suite	4:00 p.m.
	Special Entertainment	
Saturday, May 5	Past Presidents Breakfast	7:30 a.m.
	Scientific Session	8:30 a.m.-5:00 p.m.
	General Luncheon	12:00 noon
	KU Alumni Reception for Physicians & Spouses	5:30 p.m.
	Annual Presidents Banquet	7:00 p.m.
Sunday, May 6	Early Bird Breakfast	7:30 a.m.
	House of Delegates	9:00 a.m.
	Council Luncheon & Meeting	1:00 p.m.

Detailed information and registration forms will be mailed prior to the meeting.

How a Bill Becomes a Law

HON. ROBERT H. MICHEL*

Monday, April 17, 1978

Ours is a government "of the people, by the people, for the people." It is not a pure democracy. It is a republic in a democracy. It is a representative democracy.

Our laws are the embodiment of the wishes and wants, the ideas and ideals of the American people as expressed through their representatives in the Congress: 435 in the House of Representatives and 100 in the Senate. Any Member of the House or Senate may introduce a bill embodying a proposed law or revision of existing laws, at any time when his respective house is in session. When introduced the bill will be entered in the Journal of the house, and the title and sponsors of it printed in the *Congressional Record* of that day.

Each Bill Numbered

Each bill introduced is assigned a number by the Clerk of each house and referred to the Committee having jurisdiction over the subject matter by the presiding officer, i.e., the Speaker of the House or the President of the Senate. Copies of the bill are printed by the Government Printing Office and made publicly available from the Congressional document rooms.

Acting through its Chairman, the Committee decides whether a bill should be taken up by the full Committee or referred to a subcommittee for its initial consideration.

The Deliberative Stage

The Committee's deliberations are the most important stage of the legislative process. It is here that detailed study of the proposed legislation is made and where people are given the right to present their views in public hearings. When the Chairman has set a date for public hearings it is generally announced by publication in the Congressional Record.

Copies of the bill under consideration by the Committee are customarily sent to the Executive Departments or agencies concerned with the subject matter for their official views to be presented in writing or by oral testimony before the Committee. The number of witnesses, pro and con, heard by the Committee is largely dictated by the importance of the proposed legislation and degree of public interest in it.

Testimony Heard

The transcript of the testimony taken is available for inspection in the individual Committee offices. Quite frequently, dependent on the importance of the subject matter, the Committee hearings on a bill are printed and copies made available to the public.

After conclusion of the hearings the Committee proceeds to meet in Executive Sessions (sometimes referred to as "mark-up" sessions) to discuss the bill in detail and to consider such amendments as any member of the Committee may wish to offer. Each Committee has its own rules of procedure but they generally conform to the rules of the House itself.

The Committee Vote

By a formal vote of the Committee, it decides whether to report favorably to the House the bill with or without Committee amendments. A Committee report must accompany the bill, setting forth the nature of the bill and reasons for the Committee's recommended approval. The report sets forth specifically the Committee amendments and, in compliance with the rules of each house, indicates all changes the bill would make in existing law. Any Committee member, individually or jointly, may file additional supplemental or minority views to accompany the majority Committee Report. The Committee Report, accompanying the bill, is viewed by the courts and the administrative agencies as the most important document as to the intent of the Congress in the proposed legislation.

After Reporting

When a bill is reported by the Committee it is placed on the appropriate calendar. The Majority Leadership decides how and when the bill will be considered on the Floor. In general the bill is allowed to remain on the Calendar for several days to enable members to become acquainted with its provisions.

Reprinted from *Congressional Record*, proceedings of the 95th congress.

* U. S. Representative from the State of Illinois.

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In both the House and the Senate innumerable measures of relatively minor importance are disposed of by unanimous consent. In the Senate, where debate is unlimited, major bills are brought up on motion of the Majority leader and in the House are called up under a privileged resolution reported from the Rules Committee which fixes the limits of debate and whether amendments may be offered from the Floor. The Rules Committee resolution is called a "rule" for consideration of a bill: a "closed rule" if no amendments are allowed, as is generally the case in tax bills, and an "open rule" if amendments can be offered.

Reaching Consensus

While there are distinct differences between the House and Senate procedures, in general a bill is debated at length with the proponents and opponents presenting their views to acquaint the membership, as well as the general public, with the issues involved, and all with a view to arriving at the consensus. Amendments are frequently offered to make the measure more in conformity with the judgment of the majority. In the course of consideration of the bill there are various parliamentary motions, in both the House and Senate, which may be offered to determine the sentiment of the members with respect to the pending legislation. The measure may be postponed to some future date or referred back to the Committee which reported it.

With the conclusion of general debate and the reading of the bill for amendments, the question becomes whether the House or Senate, as the case may be, will pass the bill in its final form. The Congressional Record of the day the bill was under consideration will set forth the verbatim debate on the bill and the disposition made of such amendments as were offered.

After Passage

With the passage of a bill by either body it is messaged to the other with the request that they concur. If no action has been taken on the like measure by the body receiving the message the bill is usually referred to the appropriate committee of that body for consideration. Hearings are again held and the bill reported for Floor action. On relatively minor or non-controversial matters the Senate or the House accepts the measure as messaged to it by the other body.

If there are substantial differences between the House and Senate versions of a given bill, the measure is sent to a Conference Committee which is appointed by the Speaker and the President of the Senate from the ranking Committee members of each body having original jurisdiction over the bill. The object of the Conference Committee is to adjust the differences between the two bodies, and to report back to each its agreement. The report of the Conference Committee must be in writing and signed by those agreeing thereto and must have the signature of the majority of the conferees of each house.

Conference Report

The report of the Conference Committee cannot be amended and must be accepted or rejected by each house as it stands. If either house finds itself unable to accept the Conference Committee report a further conference is usually requested.

When the bill has been agreed to in identical form by both bodies a copy of the bill is enrolled, signed by the Speaker and by the President of the Senate, for presentation to the President. The bill becomes law with the President's signature of approval, or it may become law without his signature if he does not return it, with his objections, to the Congress within 10 days of its presentation to him.

If the President should return the bill, with his objections, to the originating body of the Congress, his veto may be overridden by two-thirds of both the House and Senate respectively voting to have the measure become law the President's objections to the contrary notwithstanding. Both the President's veto message and a record of the vote of the individual members in the motion to override are required by the Constitution to be set forth in the *Congressional Record*.

Council Meeting

(Continued from page 642)

It was reported that 130 physicians licenses had been revoked by the Kansas State Board of Healing Arts as of October 1, 1978. This compares to 30 revocations during 1977. The increase can probably be attributed to the CME requirements and the long reporting form needed for relicensure. The next revocation period will be after March 1979, when the 8-month extensions have expired.

Considerable problems with the EDS were reported in processing Medicaid payments. It was suggested that this be discussed by the KMS Welfare Advisory Committee and brought to the attention of the Department of SRS.

The meeting adjourned at 1:30 PM.

Writing Your Congressman

HON. MORRIS K. UDALL*

Wednesday, November 2, 1977

Surprisingly few people ever write their Congressman. Perhaps 90 per cent of our citizens live and die without ever taking pen in hand and expressing a single opinion to the man or woman who represents them in Congress — a person whose vote may decide what price they will pay for the acts of Government, either in dollars or in human lives.

This reluctance to communicate results from the typical and understandable feelings that Congressmen have no time or inclination to read their mail, that a letter probably will not be answered or answered satisfactorily, that one letter will not make any difference anyway. Based on my own 16 years' experience, and speaking for myself, I can state flatly that most of the notions are wrong. On several occasions a single, thoughtful, factually persuasive letter did change my mind or cause me to initiate a review of a previous judgment. Nearly every day my faith is renewed by one or more informative and helpful letters giving me a better understanding of the thinking of my constituents.

Mail Is Important

Mail to today's Congressman is more important than ever before. In the days of Clay, Calhoun, Webster, and Lincoln, Congressmen lived among their people for perhaps nine months of the year. Through daily contacts in a constituency of less than 50,000 people — I represent ten times that many — they could feel rather completely informed on their constituents' beliefs and feelings. Today, with the staggering problems of Government and increasingly long sessions, I must not only vote on many more issues than early-day Congressmen but I rarely get to spend more than 60 days a year in Arizona. Thus my mailbag is my best "hot line" to the people back home.

Some Fundamentals

Here are some suggestions that apply to all congressional mail:

Address it properly: "Hon. _____, House Office Building, Washington, D. C. 20515." Or "Senator _____, Senate Office Building, Washington, D. C. 20510." This may seem fundamental, but I once received a letter addressed like this: "Mr. Morris K. Udall, U. S. Senator, Capital Building, Phoenix, Arizona. Dear Congressman Rhodes. . . ."

Identify the bill or issue: About 20,000 bills are introduced in each Congress; it's important to be specific. If you write about a bill, try to give the bill number or describe it by popular title ("clean air," "minimum wage," etc.).

The letter should be timely. Sometimes a bill is out of committee, or has passed the House, before a helpful letter arrives. Inform your Congressman while there is still time to take effective action.

Write the Right People

Concentrate on your own delegation. The representative of your district and the senators of your State cast your votes in the Congress and want to know your views. However, some writers will undertake to contact all 435 Members of the House and 100 Senators, who cast votes for other districts and other States. If you happen to be acquainted personally with a Member from Nebraska, he might answer your letter, but there is a "Congressional courtesy" procedure which provides that all letters written by residents of my district to other Congressmen will simply be referred to me for reply, and vice versa.

Be Brief

Be reasonably brief: Every working day the mailman leaves some 150 or more pieces of mail at my office. Tomorrow brings another batch. All of this mail must be answered while I am studying legislation, attending committee meetings and participating in debate on the House floor. I recognize that many issues are complex, but your opinions and arguments stand a better chance of being read if they are stated as concisely as the subject matter will permit. It is not necessary that letters be typed — only that they be legible — and the form, phraseology and grammar are completely unimportant.

In the course of my years in Congress I have received every kind of mail imaginable — the tragic, the touching, the rude, the crank; insulting, persua-

* U. S. Representative from the State of Arizona.

sive, entertaining, and all the rest. I enjoy receiving mail, and I look forward to it every morning; in fact my staff people call me a "mail grabber" because I interfere with the orderly mail-opening procedures they have established. Whatever form your letter takes I will welcome it. But to make it most helpful I would suggest these "dos" and "don'ts."

Do: Write Your Own Views

Write your own views — not someone else's. A personal letter is far better than a form letter or signature on a petition. Many people will sign a petition without reading it just to avoid offending the circulator. Form letters are readily recognizable — they usually arrive in batches — and usually register the sentiments of the person or lobbying group preparing the form. I regret to report that form letters often receive form replies. Anyway, I usually know what the major lobbying groups are saying, but I don't often know of your experiences and observations, or what the proposed bill will do to and for you. And I often am not fully aware of new conditions and developments in Arizona. A sincere, well-thought-out letter from you can help fill this gap.

Do: Give Your Reasons

Give your reasons for taking a stand. Statements like "Vote against H.R. 100; I'm bitterly opposed" don't help me much. But a letter which says "I'm a small hardware dealer, and H.R. 100 will put me out of business for the following reasons . . ." tells me a lot more. Maybe I did not know all the effects of the bill, and your letter will help me understand what it means to an important segment of my constituency.

Do: Be Constructive

Be constructive. If a bill deals with a problem you admit exists, but you believe the bill is the wrong approach, tell me what the right approach is.

If you have expert knowledge, share it with your Congressman. Of all the letters pouring into a Congressman's office every morning, perhaps one in a hundred comes from a constituent who is a real expert in that subject. The opinions expressed in the others are important, and will be heeded, but this one is a real gold mine for the conscientious Member. After all, in the next 9 to 10 months I will have to vote on farm bills, transportation bills, space, health, education, housing, and veterans' bills, and a host of others. I can't possibly be an expert in all these fields; many of my constituents are experts in some of them. I welcome their advice and counsel.

Do: Say "Well Done"

Say "well done" when it's deserved. Congressmen are human, too, and they appreciate an occasional "well done" from people who believe they have done the right thing. I know I do. But even if you think I went wrong on an issue, I would welcome a letter telling me you disagreed: it may help me on another issue later.

My list of "don'ts" would include these:

Don't: Threaten or Promise

Don't make threats or promises. Congressmen usually want to do the popular thing, but this is not their only motivation. Nearly all the Members I know want, most of all, to do what is best for the country. Occasionally a letter will conclude by saying, "If you vote for this monstrous bill, I'll do everything in my power to defeat you in the next election." A writer has the privilege of making such assertions, of course, but they rarely intimidate a conscientious Member, and they may generate an adverse reaction. He would rather know why you feel so strongly. The reasons may change his mind; the threat probably won't.

Don't: Call Names

Don't berate your Congressman. You can't hope to persuade him of your position by calling him names. If you disagree with him, give reasons for your disagreement. Try to keep the dialogue open.

Don't pretend to wield vast political influence: Write your Congressman as an individual — not as a self-appointed spokesman for your neighborhood, community, or industry. Unsupported claims to political influence will only cast doubt upon the views you express.

Don't: Become a "Pen Pal"

Do not become a constant "pen pal." In a newsletter appealing for more constituent mail I do not want to discourage letters, but quality, rather than quantity, is what counts. Write again and again if you feel like it, but do not try to instruct your Congressman on every issue that comes up. And do not nag him if his votes do not match your precise thinking every time. Remember, he has to consider all his constituents and all points of view. Also, keep in mind that one of the pet peeves on Capitol Hill is the "pen pal" who weighs the mail down every few days with long tomes on every conceivable subject.

Don't: Demand Commitments

Do not demand a commitment before the facts are in. If you have written a personal letter and stated

your reasons for a particular stand, you have a right to know my present thinking on the question. But writers who "demand to know how you will vote on H.R. 100" should bear certain legislative realities in mind:

On major bills there usually are two sides to be considered, and you may have heard only one.

The bill may be 100 pages long with 20 provisions in addition to the one you wrote about, and I may be forced to vote on the bill as a whole, weighing the good with the bad.

It makes little sense to adopt a firm and unyielding position before a single witness has been heard or study made of the bill in question.

A bill rarely becomes law in the same form as introduced. It is possible that the bill you write me about you would oppose when it reached the floor.

Remember: Bills Change

The complexities of the legislative process and the way in which bills change their shape in committee is revealed by a little story from my own experience. One time a few years ago I introduced a comprehensive bill dealing with a number of matters. I was proud of it, and I had great hopes for solving several perennial problems coming before Congress. However, after major confrontations in committee and numerous amendments I found myself voting against the "Udall bill."

Finally . . .

It is now November 1977, approaching the halfway point of the 95th Congress. Before 1978 is history the House clerk will record my votes on more than 450 issues. But in a very real sense these will not be "my" votes alone: they will be yours too. There are more than 500,000 Americans in the Second Congressional District of Arizona but when the clerk calls the roll he calls only my name. Thus these votes I cast will speak for you in the decisions our country must make in the next two years.

I need your help in casting these votes. The "ballot box" is not far away. It is painted blue and it reads "U. S. Mail."

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Information for Authors

Manuscript Preparation

Manuscripts must be typewritten, double spaced, leaving wide margins. Submit the original, plus one copy if possible.

Titles should be short, specific, and amenable to indexing. A subtitle is frequently used to keep the main title short.

Summary: All manuscripts should include a short abstract which is a factual (not descriptive) summary of the work.

Author Responsibility: The author is responsible for all statements made in his work, including changes made by the copy editor. Manuscripts are received with the explicit understanding that they are not simultaneously under consideration by any other publication. Publication elsewhere will be subsequently authorized at the discretion of the Editor.

Galley Proof: To make extensive changes in the article after the text has been set in type may require an additional cost which exceeds the original. The galley proof is for correction of ERRORS, and a rewriting of the article should be done on the original copy BEFORE it is submitted for publication.

Drugs should be called by their generic names; the trade names can be added in parentheses if they are considered important. All *units of measure* must be given in the metric system.

References

Bibliographic references should not exceed 20 in number, documenting key publications. Personal communications and unpublished data should not be included. References should be arranged according to the order of citation, and not alphabetically. All references must be numbered consecutively and all must be cited in the text. Use the style of the AMA publications, giving: name of author, title of article, name of periodical, volume, pages, year.

Illustrations

All material which cannot be set in type, such as photographs, line drawings, graphs, charts, tracings (for preparation of tables, see below) must be mounted on white cardboard. All must be identified on the back as to figure number, author's name, and an arrow indicating top. Legends should be typed double spaced on a separate sheet of paper, limited to a maximum of 30 words.

Drawings and Graphs should be done professionally in India ink on illustration board or high grade white drawing paper.

Photographic material should be submitted in duplicate as high contrast, glossy prints. Color illustrations will be accepted for publication only if the author assumes the cost.

THE JOURNAL will assume the cost of B/W engravings and cuts up to $35 (or 5 cuts). Engraving cost for illustrations in excess of $35 will be billed to the author.

Tables

Because tables are set by hand, their cost is comparable to illustrations. A reasonable number of tables are allowed without cost to the author.

Tables should be self-explanatory and should supplement, not duplicate, the text. Since the purpose of a table is to compare or classify related items, the data must be logically and clearly organized. The relationship and comparison are established by the correct choice of column heads (captions of vertical columns) and stubs (left entries in horizontal listings).

Each table should be typed double spaced, including all headings, on separate sheets of lettersize paper. Oversize paper should not be used. Instead, repeat heads and stubs on a second sheet for tables requiring extra width. Number tables consecutively. Each table must have a title.

Reprints

A reprint order form with a table covering cost will be sent with the galley proof to each contributor. Since the JOURNAL has no way to provide for reprints, they must be ordered by the author and purchased directly from the printer.

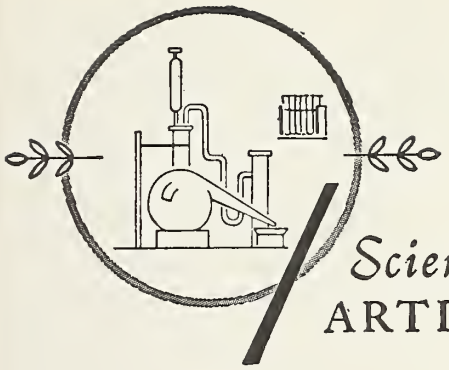


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Virus Morbidum Foetus

Congenital Malformations and Neonatal Infections

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Viral infections in pregnant women may have serious consequences. Fetal death, abortion, stillbirth, prematurity, and various forms of congenital malformation have been associated with more than ten different viruses. Maternal infection early in the first trimester (during morphogenesis) is more likely to cause congenital malformation, while infection toward the end of gestation results in neonatal disease. Moreover, maternal infection with more virulent viruses generally causes fetal death, while less virulent viruses tend to cause congenital malformation. The current knowledge concerning the role of 13 different viruses in fetal damage and neonatal disease is reviewed.

VIRAL INFECTIONS during pregnancy can be serious; abortion is common after infection with smallpox, polio, measles, and rubella viruses. Moreover, due to the endocrinologic features associated with pregnancy, the incidence and severity of certain viral diseases — *e.g.* poliomyelitis, varicella-zoster, and smallpox — are significantly increased in pregnant women. It is estimated that up to 7 per cent of liveborn babies have some form of congenital abnormality and that more than 10 per cent of these abnormalities can be attributed to maternal viral infections. Presently more than ten different viruses are known to cause intrauterine infection and fetal damage. Congenital malformation is caused by rubella and cytomegaloviruses, while herpes simplex, varicella-zoster, measles, mumps, influenza, hepatitis B, vaccinia, variola, certain echoviruses, and Coxsackie B viruses and Western equine encephalitis viruses may occasionally cause congenital malformation or severe fetal tissue damage (see below).

The greater susceptibility of the human fetus to viral infections has been attributed to the greater suitability of immature (embryonic) tissues for the replication of viruses, and the inadequacy of the immunologic response of the fetus until after the middle of the gestation period. Moreover, the placenta does not begin to function as a barrier to fetal infection until the end of the first trimester. This organ may show acute inflammation with mononuclear infiltrates and necrosis (of villous tissue) in maternal infection with rubella, herpes simplex, varicella, smallpox and vaccinia viruses, while infection with polio and Coxsackie B viruses has little discernible effect on the placenta.[1-5]

The development of congenital malformation depends on: (1) the ability of the involved virus to infect the mother and be transmitted to the fetus; (2) the stage of gestation and of morphogenesis of the fetus; and (3) the ability of the maternal virus to damage the fetus either directly by infecting it or

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indirectly through the maternal infection which alters the fetal environment adversely. Virulent viruses such as polio and smallpox viruses frequently cause fetal death and abortion, while less virulent viruses such as rubella and cytomegaloviruses tend to interfere with the normal development of the fetus and thus cause congenital malformation.

During pregnancy, maternal immunoglobulins are selectively transferred across the placenta; at birth the normal uninfected infant possesses only IgG (7S), because IgM (19S) and IgA (7-11S) do not pass the placental barrier. Recent studies have shown that the fetus is capable of synthesizing IgM immunoglobulin beginning from the fourth month of gestation. Furthermore, the synthesis of IgM will continue toward the end of gestation and is not replaced by the synthesis of IgG as is the case in postnatal life. Hence the presence of IgM in the cord or neonate serum generally indicates active synthesis of this immunoglobulin by the fetus, which in turn is induced by an antigenic stimulation (an infection). In certain congenital viral infections — *e.g.* rubella, herpes simplex, and cytomegalovirus infections — as well as in congenital toxoplasmosis, syphilis and certain other bacterial diseases, the level of IgM in the cord serum is markedly elevated. Thus the determination of IgM level in the cord or neonate serums has proved a useful tool for the recognition of intrauterine infections. However, the specific causative agent must be determined by other laboratory procedures.[6] The main viral infections of the fetus and neonate that are of particular interest are discussed in the following.

Rubella

In 1941, the Australian ophthalmologist, Sir Norman McAlister Gregg, first observed the involvement of maternal rubella in congenital malformation that was called "rubella triad" — namely, deafness, cataract, and cardiac anomalies (mainly patent ductus arteriosus). Brain damage (mental retardation, microcephaly, and hydrocephaly) was soon added to the list of the anomalies. The epidemic of 1964-65 in the United States caused birth defects in some 20,000-30,000 infants (as well as thousands of abortions and stillbirths) who, in addition to the above anomalies, showed a variety of hitherto unreported defects, such as hepatosplenomegaly, thrombocytopenic purpura, intrauterine as well as postnatal growth retardation,[7] long bone lesions, pneumonitis, jaundice, chorioretinitis, microphthalmia, encephalopathy, retarded language development,[8] diabetes mellitus,[9] hypothyroidism secondary to Hashimoto thyroiditis,[10] late onset chronic progressive panencephalitis manifested at 12-14 years of age[11] and polyneuropathy (Guillain-Barré syndrome.)[12] The term "expanded rubella syndrome" was then introduced to include all these anomalies. It has also been established that subclinical maternal infection is as deleterious to the fetus as the clinical disease, and that the virus is more teratogenic when infection occurs earlier in the course of gestation. Recent data have indicated that the average rate of malformation in infants is about 50 per cent when maternal infection occurs during the first month of pregnancy. This rate decreases to about 25, 9, and 4 per cent when maternal infection occurs during the second, third, and fourth month of pregnancy respectively. Recent serologic surveys suggest that currently about 10 per cent of pregnant women throughout the United States lack protective rubella antibodies. This ratio, however, was about 25 per cent before the epidemic of 1964. It may rise in the future if the current vaccination program is not diligently pursued. Vaccination of pregnant women, however, is contraindicated (from at least three months prior to conception) because the live virus of the vaccine can be transplacentally transmitted to the fetus.

In acquired rubella among children and young adults, the virus is present in the nasopharynx from one week before to one week after the appearance of rash, and in the blood and urine for one week preceding the rash. In such patients, antibodies develop a few days after the rash and persist for many years. The virus is eliminated from the host tissues after recovery from illness. In congenital rubella, the placenta is almost always infected, and the virus may cross this barrier and invade only certain fetal organs (causing cataract and deafness), or may disseminate throughout the body of the fetus. The congenitally infected newborns frequently continue to harbor the infectious virus (nasopharynx, urinary tract, spinal fluid) for months or years. These chronically infected children generally develop high levels of antibodies that apparently persist for life. This rules out the occurrence of immunological tolerance. However, in about ten per cent of congenitally infected babies, antibodies disappear within one to two years of age. Whether these infants are immunologically tolerant or just poor antibody producers is still debatable. The persistence of infection in these babies reflects an impairment of the mechanism involved in the recovery from viral infections. It is almost certain that antibody, phagocytosis, inflammation, and interferon do not play any role in congenital rubella. The chronic status of infection appears to indicate that certain infected clones of cells persist in the host

and continue to produce the virus. Once these cells are destroyed and other normal cells are not infected to substitute for them, the chronic infection is terminated. In the meantime, these congenitally infected babies constitute an important source of infection for susceptible individuals.

Cytomegalic Inclusion Disease

This disease is caused by cytomegalovirus which appears to be ubiquitous in distribution. It is frequently isolated from patients receiving immunosuppressive agents or having impaired immune response (*e.g.*, lymphoma patients). Furthermore, up to 1 per cent of all newborns in industrially developed countries are infected with this virus and about 10 per cent of these infected babies have gross congenital malformation. This virus is currently the most common microbial cause of brain damage in infancy. Originally, the inclusion bodies of this virus were observed at autopsy in certain enlarged cells (kidney, lung, salivary glands) that contained intranuclear and intracytoplasmic inclusions. Subsequently, many cases of cytomegalovirus infections were diagnosed in children, some of whom manifested minimal disease. It appears that maternal infection with this virus generally results in fetal infection, but congenitally infected infants infrequently manifest overt disease. In several surveys involving hundreds of pregnant women, cytomegalovirus infection, as detected by the isolation of the virus from the urine and cervix, was found in 5-28 per cent of subjects belonging to different ethnic groups. However, the rate of this infection increases with advancing gestation; in one study this rate was 1.6 per cent during the first trimester and rose to 11.3 per cent during the third trimester.[13] Another study of 1040 English and immigrant Oriental pregnant women in London showed that 42 per cent of the English and 10 per cent of the Orientals were without cytomegalovirus antibodies at the onset of their pregnancy. Among these women, 3 per cent of the first group and 16 per cent of the second experienced primary cytomegalovirus infection during pregnancy and 50 per cent of their fetuses became infected as well. However, only one-fifth of the infected babies showed mental retardation.[14] Maternal seroimmunity is not protective for the fetus, as congenital infection was seen in 3.4 per cent of offspring of 208 mothers with substantial preconceptional cytomegalovirus antibodies.[15] Mothers who become seropositive during pregnancy and excrete the virus at childbirth are at the highest risk of delivering infected infants.[16] It is estimated that three-fourths of infections during pregnancy result from reactivation of a latent virus; however, the major risk to the fetus arises from primary maternal infections. Mothers of congenitally infected babies have a cytomegalovirus-specific impairment in their cell-mediated immunity which facilitates transplacental transmission; the same type of impairment has been detected in persistently infected children of such mothers.[17, 18] Congenital defects (*e.g.*, deafness and diminished mean IQ) may be detected postnatally (months after birth) even though infection is not apparent at birth.[19] Syndromes of infantile spasms and protracted pneumonitis have been attributed to natal transmission of this virus.[20, 21] Natal infection occurs in about 5 per cent of all newborns among low socioeconomic population in the United States; however, duration and intensity of viruria and antibody response are lower in these newborns as compared to those of babies who were infected *in utero* and showed overt disease at birth.[22] The congenital malformations caused by this virus include CNS damage (microcephaly, hydrocephaly, cerebral calcification), hepatosplenomegaly, chorioretinitis, jaundice, interstitial pneumonia, thrombocytopenia, and anemia. As in congenital rubella, infants congenitally infected with this virus continue to shed the virus (in the urine and throat) for months or years, along with the continued production of antibodies. In infected adults, on the other hand, an impaired immune mechanism can be detected, but clinically serious disease is seldom observed. The virus is readily recovered from the urine and sputum of such infected adults.

Herpes Simplex Virus Infection

Presently two types of this virus are recognized. Type 1 (oral) is usually associated with lesions of the upper part of the body, *e.g.*, skin, mucous membrane, and brain; Type 2 (genital) is generally isolated from the genital area and from infants congenitally infected with this virus. The two types differ from one another in certain biologic and serologic characteristics. Intrauterine infection with this virus in infected pregnant women is well documented; ten cases of congenital malformation due to intrauterine infection have been reported. The anomalies consisted of microcephaly, intracranial calcification, diffuse brain damage, chorioretinitis, retinal dysplasia, microphthalmia, cloudy posterior lenses, psychomotor retardation, seizures, patent ductus arteriosus, and short digits. There was evidence in about half of the documented cases that the mothers had herpes progenitalis during the first trimester. Only in cardiac and digital anomalitis could the malformation be placed at the stage of em-

bryogenesis.[3, 5, 23, 24] Studies of the pathogenesis of congenital herpesvirus infection indicate that this is characteristically acquired by ascent of the virus from a resident location within the maternal cervix. The resulting chorioamnionitis with a unilateral emigration of plasma cells from fetal plate vessels to the amnionic surface of the placenta is caused by chemotaxis induced by herpes simplex virus infection.[25] Many cases of abortions have been associated with genital herpes infections during the first four months of pregnancy, but whether the virus is directly or indirectly involved is not established. Transplacentally acquired Type 1 antibodies do not protect the fetus from Type 2 virus infection.

The disease in the neonate may be the result of infection either ascending from the mother's genital tract to the fetus after premature rupture of the membranes, or acquired during the process of birth (from the infected birth canal of the mother) or shortly after (from infected mother or other individuals who come in contact with the infant). Infected neonates manifest generalized disease with fever, respiratory distress, cyanosis, and jaundice ending in death in the first few weeks of life. At autopsy, many organs (lungs, liver, spleen) are found to be involved, and typical intranuclear (Cowdry type A) inclusions are generally observed in cells bordering the necrotic areas.

Enterovirus Infections

This category includes the polio, Coxsackie and echoviruses. Poliomyelitis occurring during the first trimester is associated with a high fetal mortality. When it occurs near term, it may also be associated with a paralytic disease in the neonate. Other enteroviruses — members of Coxsackie B virus and echovirus groups — have been incriminated in intrauterine and perinatal infections. Coxsackie B virus has been isolated from stillbirths and aborted fetuses. Myocarditis and occasionally disseminated infections have been associated with Coxsackie B2, B3, and B4 viruses. Coxsackie A9 virus has been associated with digestive system defects. Recently, a right-side hemiparesis developing in a 12-week-old infant suspected of being transplacentally infected with Coxsackie B2 virus from his mother — who had symptoms of pleurodynia at 37th week of gestation — was reported.[26] Fatal generalized infection and hepatic necrosis in newborns have been associated with intrauterine infections with echovirus types 19 and 14 respectively. Echovirus type 22 has been shown to infect the placenta and be transmitted to the fetus. Most enteroviral infections, however, are acquired at birth or shortly thereafter. Neonatal infections with Coxsackie B virus (mostly types 2, 3, and 4) are generally severe and often fatal, with myocarditis as the main pathologic feature. Encephalomyocarditis and meningitis have also been observed. The echovirus (types 9 and 18) have been associated with diarrhea, rash, febrile illness, convulsions, and meningitis in the neonate. The causative virus is present in the myocardium, spinal fluid, throat, and feces.[3, 27-29]

Varicella-Zoster Virus Infection

This virus generally causes chickenpox in children (first exposure) and zoster in adults (reactivated virus or second exposure). Some ten cases of congenital malformation caused by this virus have been reported. Anomalies were cicatricial skin scarring, eye defects, and hypoplastic limbs. Severe pneumonia has also been reported. Almost all infected babies died in early infancy. Prematurity, stillbirth, or abortion have also been observed when infection occurred early in gestation.[3, 30-32]

Measles, Influenza and Mumps

The role of measles virus in congenital defects was recently investigated in ten measles epidemics (involving 368 pregnant women) which occurred in Greenland 1951-1962. The risk of abortion was up to 50 per cent when maternal infection occurred during the first trimester; this risk decreased after the first two months and became only 4 per cent for infections occurring during the second trimester. Moreover, 9 per cent of women infected during the first trimester delivered dead babies at term. Gross congenital malformations were found in 8 of 300 liveborn infants. The rate was 9 per cent for the first trimester infection (5 of 58 infants). Two cases of malformation were observed in 111 infants of mothers infected during the second trimester. One infant with congenital malformation (patent ductus arteriosus) was observed among 131 children of the third trimester infection. Malformations were atypical cerebral leucodystrophy, cyclopia with microcephaly and polydactyli, multiple cardiac anomalies (all dying within ten months), as well as anal atresia, harelip, spastic tetraplegia, stenosis of pulmonary artery, and patent ductus arteriosus (all surviving).[33] Fetal infections before term have been associated with neonatal clinical disease and giant-cell pneumonia.

Data relating fetal death, anencephaly, abortion, stillbirth, prematurity, and certain congenital malformations to maternal influenza are generally at a very low level of statistical significance. One report describes a significant increase in malformations of the central nervous system among infants of mothers

who contracted Asian influenza in 1957 during 3-9 weeks of gestation. Whether the defects were caused by the virus, the fever of the disease or the drugs (aspirin or other salicylates) taken by the mothers, is debatable.[34-36] A recent study did not support any relationship between maternal influenza and subsequent childhood leukemia and lymphoma.[37]

Increased fetal mortality has been attributed to maternal mumps; however, no virus was isolated from such fetal tissues. Mumps virus has been isolated from placentas of seronegative pregnant women who received live attenuated mumps-virus vaccine.[38] An investigation of 12 Eskimo children (aged 8-9 years), whose mothers experienced mumps during pregnancy, revealed no major physical abnormalities.[39] On the other hand, data associating intrauterine mumps virus infection with endocardial fibroelastosis are supported by experimental work in the developing chick embryo.[40]

Hepatitis, Vaccinia and Smallpox

Currently three types of viral hepatitis are recognized — hepatitis A (infectious hepatitis), hepatitis B (serum hepatitis), and hepatitis C (non-A, non-B hepatitis). Although earlier observations indicated increased maternal mortality as well as increased frequency of abortions, stillbirths, prematurity and congenital malformations in gestational hepatitis,[3] more recent studies have shown that the clinical course of maternal hepatitis is not affected by pregnancy and that the incidence of congenital malformation, stillbirth, and abortion is not increased. In a study involving 50 pregnancies complicated by acute viral hepatitis, only an overall prematurity incidence of 27.6 per cent over that of the general delivery population (10-11%) was observed; the majority of premature babies were of mothers who developed hepatitis during the third trimester. However, two infants of the eight mothers who had acute hepatitis B during the third trimester became asymptomatic carriers of hepatitis B surface antigen (HB_sAg).[41] The presence of e antigen or e antibody in the mothers plays a critical role as vertical transmission of HB_sAg is most likely to occur when e antigen (the so-called infectivity marker) is also detected in the mother. Infants of mothers with e antibody, on the other hand, normally escape hepatitis B surface antigenemia.[42] Another study showed that vertical transmission occurred in 85 per cent and 31 per cent of infants when their mothers were positive or negative for e antigen respectively.[43]

A case of transplacentally transmitted non-B hepatitis (either hepatitis A or hepatitis C) which was fatal to both the mother (nine months pregnant) and the stillborn fetus was recently reported. Viral hepatitis was documented by laboratory procedures (histopathology and biochemical tests) for both the mother and the fetus.[44]

Maternal smallpox may result in the transmission of the virus to the fetus; the outcome is commonly fetal death, abortion, or congenital disease manifesting skin lesions and pneumonia. On the other hand, vaccination of pregnant women against smallpox during the first or second trimester may result in congenital infection with vaccinia virus. The result can be fetal death, abortion, stillbirth, prematurity, or a generalized vaccinia eruption. A case of congenital vaccinia with scarring of an eyelid, choroiditis, hypoplastic bone changes, and mild skin involvement in the offspring of a mother who was vaccinated at 3-4 months' gestation has been reported.[45] At this writing, however, smallpox appears to be eradicated from the world and thus little interest remains in smallpox and vaccinia viruses.[46]

References may be obtained from the author.

CME

(Continued from page 637)

No one will be issuing a fancy CME attendance certificate or computer printout for most of the Category 2-5 educational activities. In many cases, only you will know about the educational activity.

You will therefore need to develop some sort of personal system to record your participation in these activities. I would suggest that you start keeping a CME file to note these activities as they occur — do it as soon as possible, so you don't forget. Perhaps your secretary or office manager could provide assistance to you in this area. (*Editor's Note:* She should keep the PRA application form when it comes.)

These "other categories" are in a way, a statement by AMA and others, that physicians can learn and improve their medical skills in many different ways outside the formal classroom setting. The intense reading of a pertinent journal article or interaction with colleagues can be just as educationally beneficial; in fact, it may produce the best kind of learning. Therefore, we have Categories 2-5 — no less important than Category 1 in your personal CME program.

End of commercial.

Clinical Judgment

Emergency Room Management of Patients With Chest Pain

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EMERGENCY ROOM management of the patient with chest pain has been frequently discussed, but there are little or no tangible data that clarify the value of clinical judgment in the evaluation process. The Inter-Society Commission of Heart Disease Resources has established criteria for personnel and equipment in emergency rooms as well as for coronary care units, and has established standards for transportation of patients from the emergency room to coronary care units.[1] However, when admission policy is considered, they state only that each hospital should analyze its policies for detecting and managing patients suspected of having myocardial infarction in the emergency room area, without specifying criteria for determining which patients with chest pain should be admitted and which sent for outpatient follow-up.

Fulton *et al.*, reported that 26 of 167 patients with a history of unstable angina either died suddenly or experienced an acute myocardial infarction within 3 months after onset of symptoms.[2] Of 79 sudden deaths surveyed by her group, 40 per cent of the patients had visited a physician within one month prior to the event. Of a population of patients with known coronary artery disease but no evidence of infarction who were admitted for observation and subsequently discharged, there was a 20-per cent two-year mortality rate, and 40 per cent of the deaths were sudden and unanticipated.[3] The value of electrocardiography as a diagnostic tool for determining the need for hospitalization of patients with chest pain was evaluated and found to be diagnostic in only 65 per cent of patients who subsequently were found to have myocardial infarction. In this study, the physicians' clinical judgment was found to be excellent in that all but 2 per cent of the remaining 35 per cent were admitted and few of those with noncoronary pain were admitted.[4] However, no studies have been undertaken to evaluate the role of clinical judgment in the decision making process for patients entering an emergency room for chest pain. The current study was designed to evaluate this parameter.

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Materials and Methods

In this prospective study (for three consecutive months), the records of adult patients over age 30 entering the University of Kansas Medical Center Emergency Room with chest pain were evaluated. Those with fleeting, well localized chest pain, as well as those exhibiting unequivocal electrocardiographic findings of an acute myocardial infarction, were excluded from the study. Identifying data such as name, age, sex, phone number, and address were recorded for each patient meeting the criteria for inclusion in the study. The laboratory data, disposition of the patient, follow-up recommendations, and emergency room diagnoses were recorded for each patient not admitted to the coronary care unit. The decision to admit the patient or refer for follow-up was made on the basis of clinical judgment. Because many physicians participated in this part of the evaluation, no discrete criteria were used for these determinations. Arrangements for a later, more definitive evaluation on an inpatient or outpatient basis were made through the emergency room for the majority of these patients. A nurse-clinician — utilizing telephone calls or a mailed questionnaire — attempted to obtain follow-up information on all patients evaluated, treated, and sent home from the emergency room. A subsequent check was made to

A prospective study was undertaken to evaluate clinical judgment in determining need for hospitalization of patients presenting with chest pain. Those with fleeting, well localized chest pain, as well as those exhibiting unequivocal electrocardiographic findings of an acute myocardial infarction, were excluded from the study. The findings in 241 cases demonstrate clinical judgment to be an important factor in determining the need for admission for patients presenting to an emergency room with chest pain.

determine if recommended outpatient visits and scheduled inpatient admissions were kept. During and for two months following termination of the study, local newspapers were reviewed daily for potential obituary correlation with the 180 patients sent home during the study.

Patients requiring admission to the coronary care unit were followed until discharge. Initial electrocardiographic and chest x-ray findings were recorded along with the discharge diagnoses, The results of exercise stress testing, coronary arteriography, x-ray studies, treatment, and disposition were noted. Recommended therapy and diagnostic studies were reviewed.

Results

The criteria for inclusion in this study were met by 241 patients. The mean time from the initial emergency room visit to follow-up contact was 18 days. From these 241 patients, 61 were subsequently admitted to the coronary care unit for further care and evaluation, and 180 patients were released from the emergency room. Of those released from the emergency room, 88 (49%) were contacted by telephone or questionnaire. Follow-up was not possible in 92 (51%) because of a lack of patient compliance with the questionnaire or inaccurate telephone numbers, addresses, and names given to the emergency room personnel. Of the 88 patients from whom follow-up data could be obtained, 42 had resolution of the pain and 46 continued to have pain. Eight of the latter group had an unscheduled hospitalization at KUMC or another hospital. One subsequently died of heart failure due to prosthetic valve endocarditis; three had bronchitis; two had angina; two had esophageal reflux due to hiatal hernia. There were no deaths noted in obituary columns of newspapers corresponding to names given in the emergency room.

Twenty-one patients sought further consultation with other physicians. Of the 67 patients who returned to the University of Kansas Medical Center Outpatient Department for continuation of their primary care and for treatment of their complaint, five were subsequently scheduled for admission. All five had coronary arteriograms, four of which revealed significant coronary artery disease.

Of the 180 patients released from the emergency room, follow-up recommendations for a more definitive work-up on an inpatient or outpatient basis were arranged for 140 prior to the patient's dismissal from the emergency room. Over 50 per cent of these patients did not keep scheduled clinic appointments or admission dates. Within three months following the last emergency room visit in the study, a total of 15 exercise stress tests and eight 24-hour electrocardiographic portable monitoring studies were performed post-discharge. Of the 61 patients admitted to the coronary care unit, a discharge diagnosis of pulmonary embolus was made in seven (12%), chest wall pain in four (7%), and angina in 17 (27%). Among those in the angina group, seven out of 17 had unstable angina, and six of these submitted to coronary arteriography and subsequently underwent aortocoronary saphenous vein bypass surgery. Discharge diagnoses of these 61 patients initially admitted to the coronary care unit and not mentioned above varied widely *(Table I)*.

Discussion

The population presenting with chest pain to the University of Kansas Medical Center Emergency Room was a heterogenous group that received a significant amount of its primary care in emergency rooms throughout the city or in the outpatient clinic at the Medical Center. Compliance with follow-up study was recognizably poor because of a lack of response to the questionnaire and inaccurate identifying data given to the emergency room personnel. Further, the compliance with continuing care recommendations at the time of the emergency room visit was also poor.

Lopes *et al.*, have emphasized that patients with known coronary disease who are dismissed from coronary care units and have not had an acute myocardial infarction are at high risk of sudden death.[3] They suggest a more aggressive evaluation, treatment, and follow-up program for these patients. Because the patients in this study were admitted to several different medical services within the hospital, and since many had established noncardiac diag-

TABLE I
REMAINING DISCHARGE DIAGNOSES FROM 61 CCU ADMISSIONS

Neuro-psychiatric causes	6/61 (10%)
Hypertensive cardiovascular disease	3/61 (5%)
Hiatal hernia	5/61 (8%)
Pneumonia	3/61 (5%)
Cholecystitis	2/61 (3%)
Arrhythmia	3/61 (5%)
Congestive heart failure	2/61 (3%)
Chest pain of undetermined causes	4/61 (7%)
Pericarditis	1/61 (2%)
Acute pyelonephritis	1/61 (2%)
Bronchiectasis	1/61 (2%)
Chronic active hepatitis	1/61 (2%)
Adenocarcinoma of the lung	1/61 (2%)

noses to explain the pain, a uniform plan of evaluation and follow-up was not possible.

Because hospital coronary care units and intensive care units are not prepared to accept every patient presenting to an emergency room with the complaint of chest pain, some triage is necessary and the exercise of clinical judgment is an important part of the triage. In most circumstances, a history and physical examination are sufficient to determine the need for acute hospitalization, but patients who are admitted may be found to have a variety of diseases other than heart disease.

The majority of those patients not admitted received an electrocardiogram, blood gas analysis, chest x-ray, and complete blood count; this necessitated a lengthy emergency room stay — one was documented to be three hours. Although no complications arose, parts of the emergency room work-up resulted in the patients' being away from the emergency room area to locations where the electrocardiogram could not be monitored. This was not considered ideal and even though no complications arose from this practice, it has been discontinued.

Because the emergency room is staffed by many physicians, it was difficult to assess the factors that entered into the decision making process leading to a patient's admission. However, when asked why they admitted specific patients, the most common statement by the admitting physician was, "He looked sick."

Conclusion

Patients entering the emergency room with chest pain and inconclusive electrocardiographic findings may have a variety of causes for their clinical presentation. The emergency room physician's clinical judgment is an important criterion for assessing the need for hospitalization, and is equally accurate in culling those who do not require hospitalization.

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WHAT HAS THE AMA DONE FOR ME . . .

An all too frequent question, asked primarily by those physicians least involved in the activities of organized medicine, is, "What has the AMA done for me lately?" Tom E. Nesbitt, M.D., President of the AMA, provided the answer, which was published in the September 1978 issue of the Hampden (Mass.) *Hippocrat,* and is being reprinted here in its entirety:

In my travels, I am frequently asked variations of the same question, "What has the AMA done for me lately?" by disgruntled physicians who often single out our responses to government involvement.

So I tell them what the AMA is doing, in the Congress and the courthouse, to prevent arbitrary government intervention, and to preserve the superb, healing quality of American medical education and practice.

Then I turn the tables. I ask the physician, "What have *you* done lately for the AMA, and for American medicine?" I think the question is a fair one, and deserves a fair answer. Because as physicians, we have responsibilities, as well as rights. And the best way to defend the latter is to fulfill the former, particularly our responsibilities in the socio-economic and political dimensions of medical care.

And this fulfillment, in turn, can be best achieved through the support of every physician at every level of this federation of ours, including the national level. The AMA deserves support.

I believe that each physician who graduates from a U. S. medical school owes a great debt to the AMA, which played (and continues to play) a leading role in making our medical schools the finest in the world.

I believe that each physician privileged to practice in this country owes a great debt to the AMA, which has long guarded (and continues to guard) those opportunities for medical freedom and creativity found nowhere else in the world.

Finally, I believe that each physician who fails to support the AMA is failing to live up to those traditions and principles upon which the past, present, and ultimately the future, of American medicine rest.

Such physicians, the "free riders" of medicine, might heed the words of the late Adlai E. Stevenson, the American attorney and diplomat, who said, ". . . it is often easier to fight for principles than to live up to them."

Carcinoid Heart Disease

Restrictive Cardiomyopathy as a Late Complication

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CARCINOID heart disease occurs in more than one-half of patients with malignant carcinoid tumors with metastases.[1] Most of these cases have pulmonic and tricuspid valve lesions, and right ventricular failure is a leading cause of death. The patient in this report survived 22 months after pulmonic and tricuspid valve replacement and developed findings of both right and left ventricular failure.

Case Report

A 24-year-old female was admitted to Grady Memorial Hospital, Atlanta, Georgia in July 1973, for evaluation of flushing episodes, recurrent diarrhea, and progressive dyspnea on exertion. Physical examination revealed acrocyanosis and auscultatory findings of pulmonic stenosis and tricuspid regurgitation. Urinary 5-hydroxy-indoleacetic acid excretion was 111 mg/24 hrs (normal: 2-8 mg/24 hrs).

The patient moved to the Kansas City area and was admitted to the Kansas University Medical Center because of progressing symptoms. In addition to previous findings, peripheral edema and hepatomegaly were present, but there were no signs of left ventricular failure. Chest roentgenogram was normal, and the electrocardiogram showed right axis deviation. (The data from the right heart catheterization are listed in *Table I.*) Because her functional capacity was markedly impaired, tricuspid and pulmonic valve replacement with porcine heterograft prosthetic valves was performed in October 1973. Open liver biopsy at this operation confirmed the presence of metastatic carcinoid lesions.

Postoperatively, the patient's dyspnea, hepatomegaly, and peripheral edema improved for several weeks but then worsened again. Cardiac catheterization was repeated 13 months postoperatively to evaluate prosthetic valve function and to search for other potentially remediable lesions. Retrograde left heart catheterization was included, and the pressure data are included in *Table I.* Right ventricular and pulmonary angiography confirmed the adequacy of function of the prosthetic valves, with no regurgitation and only a small systolic gradient across the pulmonic prosthesis. The right ventricle was seen to be moderately dilated and poorly contractile. Both right and left ventricular pressure curves show prominent dip-and-plateau patterns in diastole with elevated but unequal end-diastolic pressures. The right atrial pressure curve exhibited a marked "y" descent of 16 mm Hg *(Figure 1).* Left ventricular ejection was 50 per cent and there were no hypokinetic segments of the myocardium. During left ventricular angiography the coronary arteries were seen on the very surface of the cardiac shadow, with no evidence of a thickened pericardial peel. Myocardial thickness was normal.

A patient with carcinoid heart disease had pulmonic and tricuspid valve replacement after developing right ventricular failure. More than one year later, clinical and catheterization findings of a restrictive cardiomyopathy were presumed to be related to extensive endocardial plaque formation. This complication probably represents the extended natural history of this disease.

After hospitalization, the patient was treated with digitalis and diuretics, but never attained more than a

TABLE I

*Parameter**	*Preoperative 10-8-73*	*Postoperative 11-27-74*
Cardiac index	-	1.9
Right atrium	20	22
Right ventricle	56/16	80/25
Pulmonary artery	24/8	60/25
Pulmonary wedge	8	28
Left ventricle	-	95/0-30
Aorta	-	95/70

* Cardiac index in liters per minute per M^2. All pressures are in mm Hg.

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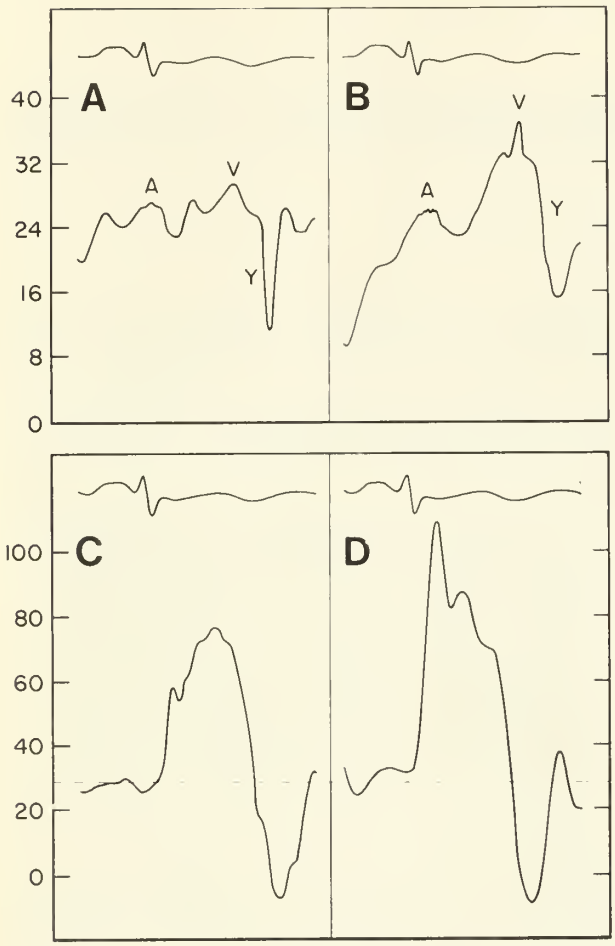


Figure 1. Pressure tracings obtained during postoperative catheterization: A — Right atrium, B — Pulmonary capillary wedge, C — Right ventricle, D — Left ventricle. All pressures are in mm Hg.

marginal activity tolerance (Class III-C), and she died in August 1975. An autopsy was not performed.

Discussion

The majority of reports of patients with cardiac metastases from carcinoid tumors have demonstrated right-side valve lesions, especially tricuspid regurgitation and pulmonic stenosis.[2] These lesions are often directly associated with the patient's gradual deterioration and death, even when the primary tumor has been surgically removed.[2, 3] Left-side lesions occur in a significant number of cases, but have not been as severe as those on the right.[3]

Patients having once developed right ventricular failure may die prior to the appearance or progression of left-side lesions. Our patient had adequately functioning tricuspid and pulmonic valves and survived 22 months after operation and 25 months after diagnosis.

Several mechanisms could have produced the patient's clinical picture, including constrictive pericarditis or pericardial effusion, myocardial disease or restriction of ventricular motion by extensive formation of endocardial plaques in both right and left ventricles:

1. Carcinoid pericarditis has been known to occur even in the absence of valvular lesions, and may be associated with pericardial effusion.[4] The appearance of the left ventriculogram showed no evidence of pericardial thickening, and the right and left ventricular diastolic pressures were unequal, so that external constriction of the ventricles is unlikely.

2. Congestive and hypertrophic cardiomyopathies were ruled out because of the relatively normal appearance of the left ventricular contraction and myocardial thickness, as well as the classic dip-and-plateau pattern of the right and left ventricular pressures. Antineoplastic chemotherapy was given for only a very short period of time because of poor patient compliance. At no time did she receive methysergide or other drugs known to produce nonspecific tissue fibrosis.

3. Several lines of evidence make restrictive cardiomyopathy the most likely diagnosis. Pericardial thickening was absent, right ventricular contractility was poor, while that of the left ventricle was less than would be expected in constrictive pericarditis. Pulmonary arterial hypertension of this degree is unusual in a chronic constrictive process, as is the inequality of the ventricular end-diastolic pressures.[5] Mean left atrial pressure was elevated above that of the right, whereas in a constrictive pattern both atrial pressures tend to be equal. Restriction of ventricular motion would occur with heavy plaque formation on the ventricular endocardium, producing mechanical impairment of contraction and relaxation. Roberts *et al.* reported such a mechanism in several cases with nondistensible right atria.[6] Postmortem descriptions of patients with left-side lesions have emphasized the relative sparing of left ventricular endocardium as opposed to the right; however, the added life span afforded our patient by valve replacement would have provided opportunity for more extensive development of endocardial lesions.

Surgical relief of right-side valvular dysfunction has been performed in other instances with variable success.[7-9] The complication described in this report would represent a serious limitation to any improvement in prognosis expected from surgery.

(Continued on page 665)

Catecholamine-Secreting Ganglioneuroma

A Surgically Correctable Cause of Hypertension

JOHN ADAMS, M.D.; DONALD MOELLER, M.D.; THOMAS ZINN, M.D. *and* **ALAN ROTH, M.D.,** *Kansas City, Kansas*

HYPERTENSION associated with elevated catecholamine metabolite levels in the serum or urine of the adult patient is usually indicative of pheochromocytoma. However, other neural crest neoplasms — including neuroblastomas, ganglioneuroblastomas, and ganglioneuromas — are also capable of catecholamine elaboration.[1] Reported is a case of a catecholamine-producing, retroperitoneal ganglioneuroma in a 64-year-old female with hypertension.

Case Report

A 64-year-old white female with a known history of colonic diverticulosis was admitted to the hospital for evaluation of hematochezia. There was no previous history of melena, diarrhea, hypertension, headaches, dizziness or palpitations, but the patient reported having experienced occasional flushing episodes in the recent past. Physical examination revealed blood pressure 170/100 mm Hg; other findings were normal. A quantitative 24-hour urine collection showed an elevation of vanilmandelic acid (VMA) to 116.3 mg. Colonoscopy confirmed the diagnosis of diverticulosis of the sigmoid colon. The patient was discharged after spontaneous resolution of rectal bleeding, but was readmitted three weeks later for further evaluation of persistent hypertension in the range of 150/90-180/110 mm Hg. Again the 24-hour urine collection demonstrated elevations of VMA, metanephrines, and catecholamines *(Table I)*. Intravenous pyelography yielded normal results, but selective right renal arteriography revealed a 6x10 mm tumor blush in the right suprarenal region *(Figure 1)*.

Reported is a case of a catecholamine-secreting ganglioneuroma in a hypertensive 64-year-old female. Surgical removal of the tumor resulted in marked reduction of urinary catecholamine metabolite excretion and return to a normotensive state. Although ganglioneuroma is a rare neoplasm in the adult patient, it should be considered along with pheochromocytoma as a surgically correctable cause of hypertension.

Laparotomy was performed three months later, and a 10x17x6 mm well circumscribed grayish-tan mass was removed from the paravertebral region on the right side. Microscopy showed histological features characteristic of a ganglioneuroma *(Figure 2)*. Mature ganglion cells, appearing singly and in aggregates, were easily identified. The ganglion cells were interspersed in an organized fascicular

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TABLE I
SERIAL 24-HOUR URINE AND MEAN ARTERIAL BLOOD PRESSURE VALUES

Date	*8/75*	*9/75*	*10/75*	*1/76*	*10/76*
Vanilmandelic acid (0-10.0 mg/TV)*	116.3	11.8	26.7		10.5
Metanephrines (0.3-0.9 mg/TV)*	—	1.7	—		0.4
Catecholamines (0-115 *m*g/TV)*	—	163	—		70
Mean arterial blood pressure	133	123	—	↑ Ganglioneuroma resected	100

* Normal values for a 24-hour quantitative urine collection.

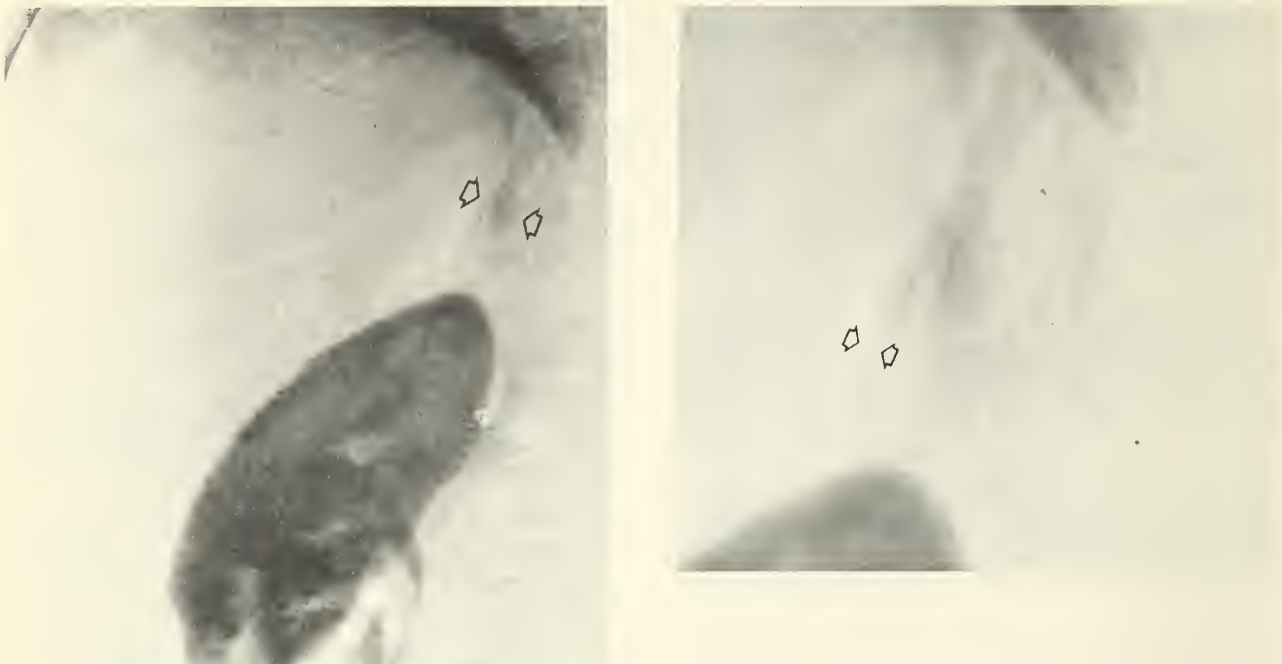


Figure 1. Left, subtraction tomography of a selective right renal arteriogram showing a 1-cm tumor blush in the right suprarenal region (arrows). Right, magnification of the region showing the tumor and its draining vein (arrows).

network of Schwann cells with wavy, spindle-shaped nuclei and cytoplasmic fibrils. Microscopy of the right adrenal gland removed at surgery was normal.

Eight months following surgical resection of the neoplasm, the patient demonstrated a decrease in mean arterial blood pressure as well as a marked reduction in urinary excretion of VMA, metanephrines, and catecholamines *(Table I)*.

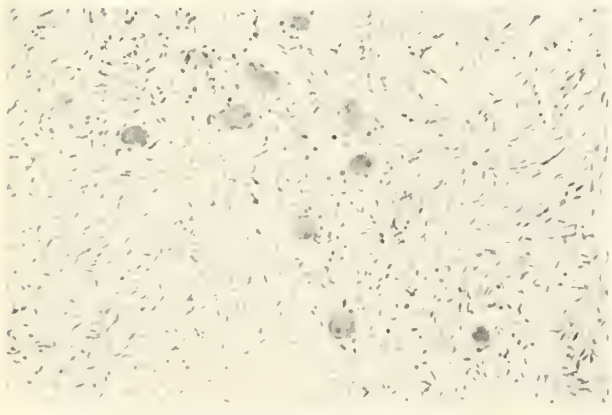


Figure 2. Tumor section showing mature ganglion cells interspersed in a fascicular network of Schwann cells.

Comment

Ganglioneuromas are slow growing neoplasms composed of nerve fibers and mature ganglion cells.[2] These tumors are the most benign of the primitive neuroectodermal tumors, with the more malignant, less well-differentiated ganglioneuroblastoma and neuroblastoma lying at the opposite end of the pathologic spectrum. According to several authors,[3-6] ganglioneuromas are more commonly found in females, and 60 per cent of the patients are under the age of 20. In a large review of 417 cases of ganglioneuroma,[6] 43 per cent occurred in the mediastinum, 32 per cent in the abdomen, and 8 per cent in the neck, with the majority of the tumors originating in or near the autonomic nervous system ganglions. Although there have been reports of local invasion and lymph node metastases, as a rule these neoplasms remain well localized. The majority of patients harboring ganglioneuromas are asymptomatic, but they may present with hypertension, headache, palpitations, episodic diaphoresis and

flushing, and with diarrhea if the tumor is hormonally active. The treatment of choice is surgical excision.[7]

As was the case with our patient, histological examination of the tumor demonstrates Schwann cell cords arranged in interlacing fascicles. Mature ganglion cells with their characteristic abundant pink cytoplasm and prominent nucleoli are irregularly interspersed or arranged in compact clusters within the Schwann cell matrix. Disarray and the presence of vacuoles within the fascicles signal immaturity, so all ganglioneuromas must be carefully examined to exclude the presence of a malignant lesion.

The patient described in this report demonstrated several unusual manifestations of ganglioneuroma. She was 64 years old at the time of diagnosis, while the majority of ganglioneuromas occur in the first two decades of life. Hassenbusch,[7] however, has described this type of tumor in a 71-year-old patient. The characteristic arteriographic picture of a large vascular mass[8] was not present in this case. By arteriography this patient's tumor appeared to be less than 1 cm in diameter with a paucity of neovascularity. Despite its small size, this ganglioneuroma produced significant amounts of vasoactive amines and subsequent hypertension *(Table I)*. As with pheochromocytomas, there appears to be little relationship between the size of the tumor and the quantity of catecholamine elaborated. Moreover, in this case, surgical removal of the ganglioneuroma normalized the patient's blood pressure and reduced the excretion of urinary VMA, metanephrines, and catecholamines *(Table I)*.

Summary

The case presented involved a metabolically active ganglioneuroma in a hypertensive 64-year-old female. Surgical excision of the tumor resulted in return of the patient to the normotensive state and a marked reduction in the urinary excretion of catecholamine metabolites. Although ganglioneuroma is a rare neoplasm, it should be considered with pheochromocytoma as a surgically correctable cause of hypertension in the adult patient.

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Carcinoid Heart Disease

(Continued from page 662)

Summary

In this young woman with carcinoid heart disease, clinical and hemodynamic findings of restrictive heart disease developed 13 months after pulmonic and tricuspid valve replacement. Several possible explanations were considered, but the data included in this report are most compatible with extensive endocardial deposition of carcinoid plaques, resulting in a poorly compliant right ventricle. This complication probably represents the extended natural history of this disease in patients whose right-side valvular lesions are repaired.

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Atrial Flutter

Termination by Rapid Atrial Pacing

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ATRIAL FLUTTER often presents a difficult therapeutic problem. Although this arrhythmia may be paroxysmal and transient, it is frequently persistent and may result in compromised hemodynamic function in patients with heart disease. Loss of effective atrial contraction and rapid ventricular response may reduce cardiac output and cause heart failure or syncope. Rapid ventricular rate may increase myocardial work, decrease coronary filling, and result in angina. Treatment with digitalis and antiarrhythmic drugs frequently fails to accomplish adequate control of the ventricular rate or conversion to sinus rhythm. Although synchronized direct current (DC) cardioversion may effectively correct the arrhythmia, many patients have received digitalis, and with digitalis excess there is increased risk of ventricular arrhythmias.[1, 2] Furthermore, the required anesthesia or amnestic dose of intravenous diazepam are a hazard in patients with severe obstructive lung disease, who frequently develop atrial flutter. Sick sinus syndrome represents an additional contraindication to DC cardioversion.[3] Recent thoracotomy, recent mastectomy, fear, and anxiety may also present practical problems for performing DC cardioversion. Rapid atrial pacing has been used recently with varying success to control atrial flutter.[4-11] This paper presents our experience with the technique and some considerations regarding its place in the treatment of atrial flutter.

Atrial flutter may present a difficult therapeutic problem in control of the ventricular rate and compromised hemodynamic function. Synchronized direct current cardioversion is effective, but may be hazardous in patients with digitalis toxicity, severe obstructive pulmonary disease (because of the need for sedation) and sick sinus syndrome. Rapid atrial pacing offers a useful alternative to DC cardioversion in these settings.

Patient Selection

Rapid atrial pacing was performed 11 times in ten patients. All ten patients had cardiac disease: six had arteriosclerotic heart disease; two had rheumatic heart disease with predominant mitral involvement; one had surgically corrected aortic insufficiency; one had Wolff-Parkinson-White syndrome, myocardiopathy of unknown cause, and sick sinus syndrome.

Additional complicating factors were present in six patients: three had severe congestive heart failure; two had severe obstructive lung disease; one had bacterial pneumonia; one had chronic renal failure with uremic pericarditis; one was postoperative from a radical mastectomy; one had symptomatic carotid arterial obstruction. Nine of the ten were receiving digitalis, and one was suspected of digitalis intoxication. All patients were receiving antiarrhythmic drugs (7 quinidine, 2 procainamide, 1 propranolol). Duration of atrial flutter ranged from 2-14 days, except for one patient whose flutter had been chronic.

Diagnosis

Atrial flutter was diagnosed by the usual criteria of a regular atrial rate of 250-350/min with a saw tooth baseline in standard electrocardiographic leads II, III, and V_1. The atrial rate slowed to 230/min in several cases with quinidine therapy, but the saw tooth pattern persisted.

Procedure

Rapid atrial pacing was performed either in the cardiac catheterization laboratory or at the bedside in the intensive care unit employing a portable image intensifier. Using local anesthesia, a bipolar pacemaker catheter was introduced through an antecubital vein cutdown or percutaneously from the femoral vein. The catheter was advanced under

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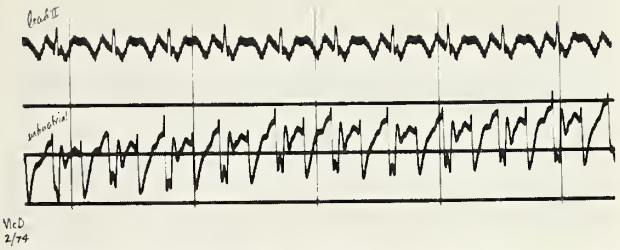


Figure 1. Lead II and intra-atrial lead at 50mm/sec characteristic of atrial flutter with 2:1 AV block.

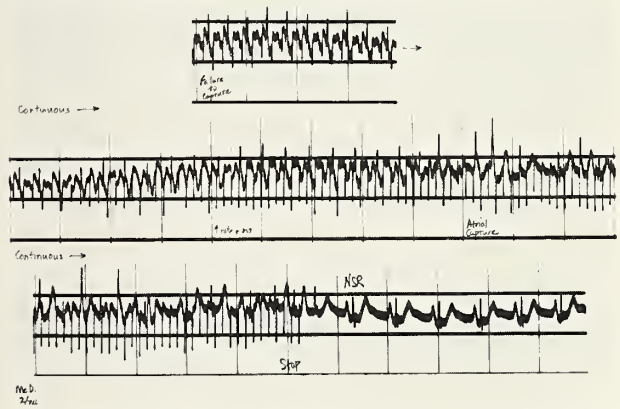


Figure 2. Intra-atrial lead from the same patient with atrial stimulation at 350/min increased to 450/min with atrial capture and resultant conversion to sinus rhythm clearly revealed when atrial stimulation is terminated.

fluoroscopic control into a stable position at the lateral wall of the right atrium. Using a battery powered external pacemaker (Medtronic Model 1397) the atrium was stimulated at rates up to 500/min with a pulse duration of 2 msec and an output of 3-15 ma. Position was checked prior to pacing by fluoroscopy, and in some instances also by intra-atrial electrocardiogram. In most cases stimulation was performed first at a rate of 100/min and then gradually increased until atrial capture was accomplished.

Results

Rapid atrial pacing terminated atrial flutter in eight of eleven attempts. There were seven conversions to sinus rhythm and one to atrial fibrillation. All six patients with arteriosclerotic heart disease converted to sinus rhythm. One patient with rheumatic heart disease converted to atrial fibrillation; the other failed to convert. The patient with surgically corrected aortic insufficiency converted to sinus rhythm on one occasion, but failed to convert from another episode six months later. Rapid atrial pacing was unsuccessful in the patient with Wolff-Parkinson-White syndrome, cardiomyopathy, and sick sinus syndrome. All of the patients with severe

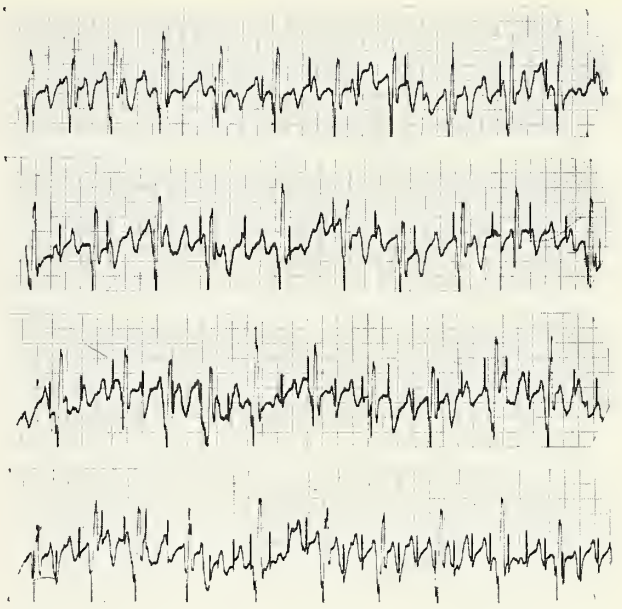


Figure 3. MCL_1 monitor lead with atrial flutter and atrial pacing begun at 100/min and gradually increased in rate, capturing the atrium.

heart failure converted as did all those with complicating disorders.

In the eight conversions only one occurred at a pacing rate slower than the flutter rate. Atrial fibrillation was produced in two patients. It was persistent in one of the patients with rheumatic heart disease, but it immediately reverted to atrial flutter in the other patient and repeat stimulation produced sinus rhythm. The remainder converted directly to sinus rhythm.

Several representative cases are reviewed. *Figure 1* is from a patient with arteriosclerotic heart disease and severe obstructive lung disease, and shows an intra-atrial lead and lead II electrocardiogram recorded at 50 mm/sec prior to pacing. In *Figure 2,* atrial stimulation is begun, initially at 350/min, then at 450/min with atrial capture. In the right side of the middle tracing, the contour and rate of the atrial complexes change indicating cessation of flutter. Sinus rhythm supervenes with the termination of pacing.

Figure 3 is an MCL_1 monitor lead from a patient with arteriosclerotic heart disease, severe congestive heart failure, and digitalis intoxication. Pacing is begun at a rate of 100/min and gradually increased. In the bottom row, capture occurs with modification of the atrial waveform. In *Figure 4,* a discreet "p" wave is produced by the pacemaker and no further flutter activity is apparent. After stopping atrial stimulation there is a brief pause, a sinus escape, and then a stable sinus rhythm.

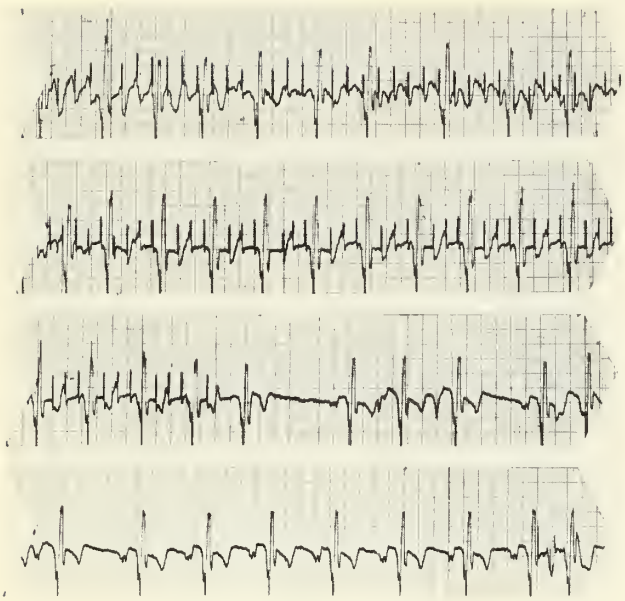


Figure 4. Continuous MCL_1 in the same patient reveals cessation of flutter activity in the second row. After terminating the atrial stimulation, sinus rhythm occurs.

The last case is of a patient with rheumatic heart disease. *Figure 5* displays a surface and an intra-atrial electrocardiogram showing atrial flutter with a 2:1 ventricular response. In *Figure 6* rapid atrial pacing immediately produces a more rapid atrial rate and a slower ventricular response. After an 8-second period of stimulation the rhythm converts to atrial fibrillation.

Four of the eight patients maintained sinus rhythm for an indefinite period. The patient who converted to atrial fibrillation remains in that rhythm. Three patients reverted to atrial flutter at three months, twelve days, and nine days respectively. The last mentioned patient had chronic atrial flutter and also failed to maintain sinus rhythm after a subsequent DC cardioversion.

Discussion

The electrophysiologic mechanism of atrial flutter remains an undecided issue between the ectopic focus, or automatic concept,[12] and the "circus movement" or re-entry concept.[13] Termination of atrial flutter with atrial pacing has been attributed to three conceptual mechanisms: (1) interruption of a circus or re-entry pathway (by rendering a partially refractory area completely refractory);[6-8, 11] (2) overdrive of an ectopic focus with post pacing sinus recovery;[5, 12] and (3) creation of a new unstable atrial rhythm from which conversion to sinus rhythm occurs.[5-8, 12]

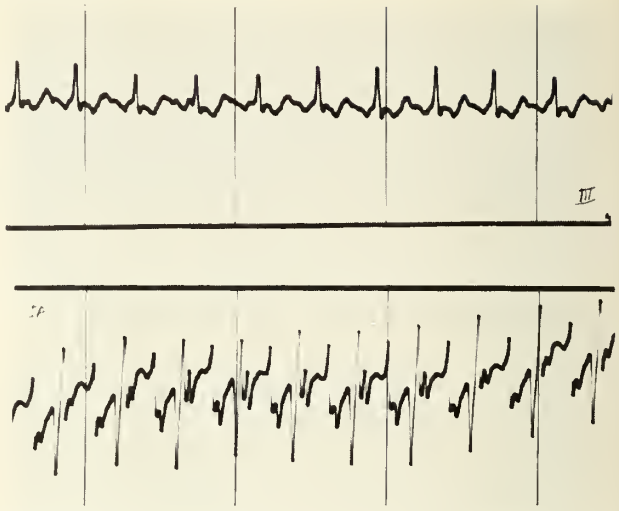


Figure 5. Lead III and intra-atrial lead show atrial flutter with 2:1 AV block.

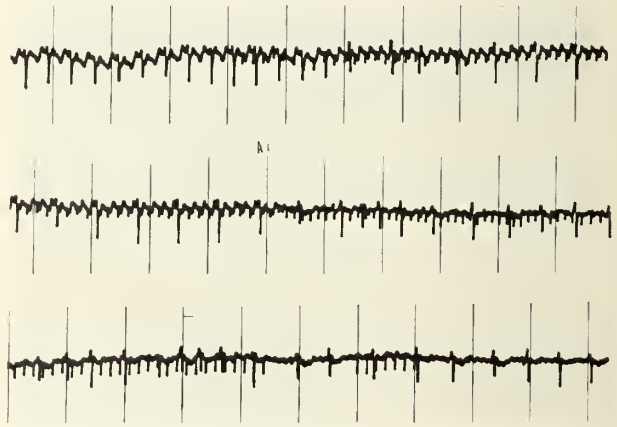


Figure 6. Intra-atrial lead from the same patient demonstrates rapid atrial pacing producing a more rapid atrial rate and slower ventricular response. When atrial stimulation is terminated, conversion to atrial fibrillation is revealed.

In the present series of patients, one converted to sinus rhythm with pacing at a rate slower than the flutter rate suggesting interruption of a re-entry pathway. Five conversions to sinus rhythm occurred after pacing at a rate faster than the flutter rate and were thus consistent with any of the three concepts. Atrial fibrillation was induced in two cases. These results suggest that all three of the mechanisms were involved.

In all reported series of rapid atrial pacing for atrial flutter,[5-11] the conversion rate ranges from 0-70 per cent to sinus rhythm and from 40-95 per cent to either atrial fibrillation or sinus rhythm. Our results are comparable. These results indicate the efficacy of this treatment.

Although conversion rates with DC cardioversion are higher and more consistent,[14] in four of our eleven patients DC cardioversion was contraindicated due to digitalis intoxication, sick sinus syndrome, or the danger of sedation. Clearly rapid atrial pacing does not supplant DC cardioversion in treatment of resistant atrial flutter, but it does offer an alternative in certain high risk patients.

Conversion to atrial fibrillation is often helpful, since atrial fibrillation nearly always has a slower ventricular rate than atrial flutter and can be more satisfactorily managed with drug therapy. As a result, paced induction of atrial fibrillation has been used by several investigators to treat intractable atrial arrhythmias.[15, 16]

Summary

Although atrial flutter is frequently a transient arrhythmia, it may be persistent and present a difficult therapeutic problem in control of the ventricular rate and compromised hemodynamic function. Synchronized direct current cardioversion is effective, but may be hazardous in patients with digitalis toxicity, severe obstructive pulmonary disease (because of the need for sedation), and sick sinus syndrome.

Rapid atrial pacing successfully terminated eight of eleven episodes of atrial flutter in ten patients. Although all of the above relative contraindications to DC cardioversion were represented, no complications were encountered.

It appears that rapid atrial pacing offers a useful alternative to DC cardioversion in the clinical settings of severe obstructive lung disease, digitalis intoxication, and sick sinus syndrome.

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Transient Global Amnesia

Report of a Case Secondary to Bilateral Middle Cerebral Artery Involvement

I. AHMED, M.D.,* *Leavenworth*

Transient global amnesia (TGA) is a well recognized clinical entity. Previous studies have suggested involvement of vertebrobasilar system. Presented is a patient with an episode of transient global amnesia secondary to bilateral middle cerebral artery involvement with complete occlusion on one side and temporal branch occlusion on the other side. In the present study, with the help of computerized axial tomography scan, it was possible to demonstrate that the lesion in TGA is infarction rather than temporal lobe seizure as has been suggested by some authors in the past.

TRANSIENT GLOBAL AMNESIA syndrome may be defined as a sudden episode of loss of memory for recent events and inability to recall recently learned information associated with retrograde amnesia and variable automatism.

Miller-Fischer and Adams[1] described in detail 17 patients with this syndrome. These authors consider temporal lobar seizure or ischemia as possible causative factors. Several reports[2, 6] have appeared in literature since then and ischemic causes have been favored especially in the vertebrobasilar system. Other factors contributing to atherosclerosis have been emphasized.[4] Presented here is a case of transient global amnesia secondary to involvement of middle cerebral artery bilaterally, complete occlusion on one side, and temporal branch occlusions on the other side. To our knowledge, no such report has appeared in literature for which the implicating defect is anterior circulation resulting in TGA syndrome, without any other associated neurological signs and symptoms, or any evidence of posterior circulation involvement.

Case Report

A 53-year-old white male was admitted to Neurology Service because of an episode in which, while on the job and seemingly oriented, he could not remember his colleagues and friends, wandered in the office, and repeatedly asked the same question of everyone as to where he was. This lasted for 15-20 minutes, following which the patient was normal but had no memory of the episode of bizarre behavior. On admission to the hospital the only neurological deficit present was amnesia for the period during which he was behaving strangely. The only significant factor in his medical history was an episode occurring 14 years earlier. While in the armed service, the patient developed a transient weakness of the right side of the body that terminated after several hours duration. He was told his circulation was poor, but received no treatment; no neurological deficit was manifest during the ensuing years. The patient was on active duty until his retirement two years prior to this admission.

Physical examination revealed: blood pressure, 130/80 mm Hg; pulse rate, 72/min; respirations, 18/min. Findings of a general physical examination were within normal limits, as were those of neurological examination except for the persistent amnesia for the episode of bizarre behavior. CBC and serum chemistries were within normal limits. Electroencephalogram revealed a persistent 2-5 CPC slow activity in the right side diffusely, but this was not seen on subsequent EEGs. Computerized axial tomography scan showed a small area of reduced density on the right posterior temporal region; this was consistent with infarction in that area *(Figure 1)*. Cerebral angiography revealed complete occlusion of left MCA *(Figure 2)*; occlusion of temporal branches of the middle cerebral artery on the right side *(Figure 3)*, and normal vertebrobasilar circulation *(Figure 4)*. No other abnormalities were noted.

The patient had suffered transient ischemic episode 14 years earlier involving the left carotid arterial system. Since that time he had completed active duty in the Army and was engaged in full-time employment enjoying normal health until the current

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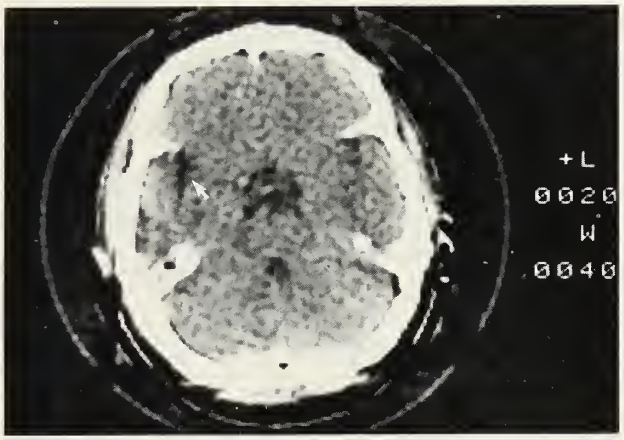


Figure 1. Photograph of CAT scan. Arrow indicates area of infarction.



Figure 2. Photograph of left carotid angiogram showing complete occlusion of the left middle cerebral artery (arrow).



Figure 3. Photograph of right carotid angiogram showing temporal branch occlusion of the right middle cerebral artery.

episode. There was no demonstrable neurological deficit except in the sphere of memory.

Discussion

For clinical purposes, memory may be divisible into three subdivisions:[7, 8] (1) immediate memory characterized by widespread neural activity lasting a few seconds to one minute; (2) short term memory involving transduction of neural activity into macromolecular form continuing minutes to one-half hour; and (3) long term and remote memory dependent on widespread storage of macromolecular engrams lasting perhaps a lifetime. The biochemical concept of neuronal memory has been described for the above stages. According to clinical studies of Symonds,[9] the neural substrate for memory consists of memory units registered in a vast number of neurons. As in all biological systems, this storage has a tendency to decay, but it is believed to be reinforced by the limbic system which functions as a



Figure 4. Photograph of vertebral angiogram showing normal posterior circulation.

strong activating system. If there is damage to this system, the ability to store new memory is lost and stored memory decays. Accordingly, recent memory is first to be lost because it is reinforced the least. If this hypothesis is true then it is reasonable to conclude that TGA results from temporary inactivation of the limbic system resulting in a significant defect in second stage memory. Anatomically, lesions have been demonstrated in the hippocampal formation bilaterally.[10]

Commonly, TGA is seen with haemodynamic changes in the vertebrobasilar system because it affects the temporal lobe bilaterally.

In the present case, the patient had a middle cerebral artery occlusion on the left side and occlusion of the temporal branches on the other side producing ischemia infarction on the right side with possible ischemia on the other side. CAT scan clearly demonstrated infarction on the right side in the temporal area that — combined with some ischemia on the opposite side — could have produced the syndrome of TGA. In this study, CAT scan findings support the view that the causes of TGA are vascular in nature, rather than seizure discharges as had been suggested in some prior studies.

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Current COMMENT

The Hirsute Female

TIMOTHY O. WAHL, M.D.,* ***and***
JOSEPH L. KYNER, M.D., † ***Kansas City, Kansas***

EXCESS BODY HAIR in the female may be a frustrating problem not only to the patient but to the physician attempting to find a treatable cause for this common complaint. Indeed, it may be the most common potential endocrine problem in the female.

Body hair socially acceptable in one society is not in another. Racial, ethnic and familial factors, aging, physiologic responses in pregnancy, and obesity are all elements that may account for an increase of hair in the otherwise normal woman. However, as will be demonstrated, it is important that the possibility of an endocrine disorder not be overlooked. This is particularly so if another problem such as amenorrhea, menstrual irregularity, or virilization is present. With any of these combinations, however, the physician usually feels justified in pursuing a workup. More often than not, the physician's problem is how far to go or what to do with the complaint of just plain "hairiness" in the borderline to mildly hirsute patient.

Type of Hair

There are two types of hair. Vellus hair is soft, unmedullated, only occasionally pigmented, usually shorter than 2 cm, and accounts for most body hair. Terminal hair is coarse, always pigmented, longer than vellus hair, and — except for scalp, eyebrows, and eyelashes — normally makes its appearance at puberty. The difference in the amount or location of vellus versus terminal hair is the response to androgen. Hair has varying thresholds of androgen response in different areas of the body (pubis the lowest, face the highest). Therefore, the presence of excess facial hair may be more suggestive of a hormonal problem than would be excess pubic hair.

Endocrine vs Normal Hair

The question of endocrinologically significant hirsutism arises when otherwise normal-for-pubescence axillary and pubic hair arises in the child, or when normal-for-the-adult-male terminal hair presents in the female. The endocrinologist views hirsutism as a physical finding suggestive of the presence of excessive quantities of androgenic hormone activity. Although the traditional definition of hirsutism as the overgrowth of hair of any sort in unusual locations or in greater abundance than usual is of little use clinically, it is most important to the concerned, body-conscious patient.

Classification

The differential diagnosis of hirsutism is outlined in *Table 1*. The most important points to consider are the racial and familial parameters. For example, individuals of Mediterranean ancestry are more likely to have excess hair and will present a history of ancestral females with the same problem.

The pathologic conditions that cause hirsutism are rare. In most cases, the physician is obligated to consider an endocrine disorder because, if present, it may be easily treated with relatively good results.

The adrenal gland may be responsible for hirsutism in only a few people with this clinical problem. Both congenital adrenal hyperplasia and adult adrenogenital syndrome are enzymatic errors in

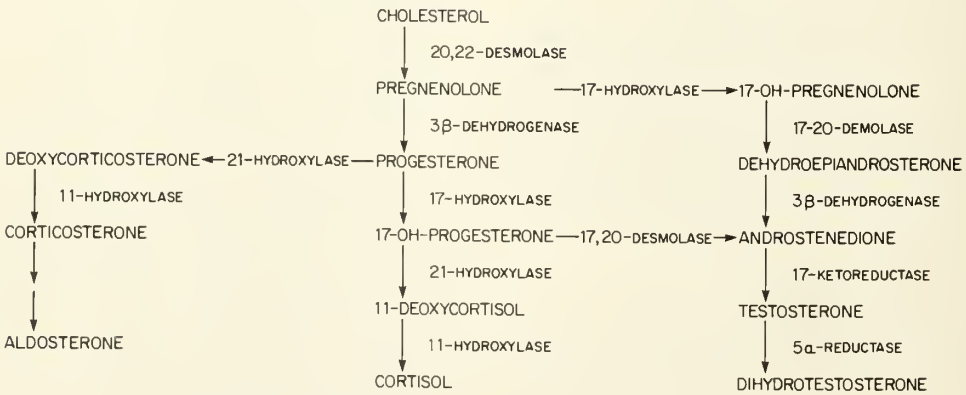
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TABLE I
CLASSIFICATION OF HIRSUTISM

I. Normal subjects (without virilization)
  A. Intrinsic
    1. Genetic (racial or familial)
    2. Physiologic
      a. pregnancy
      b. menopause
    3. Idiopathic
  B. Extrinsic
    1. Trauma
    2. Drugs (Diphenylhydantoin, corticosteroids, androgens)
    3. Infections (encephalitis)
  C. Skin lesions
II. Pathologic conditions (usually with virilization)
  A. Endocrine system
    1. Adrenal
      a. congenital adrenal hyperplasia
      b. adult adrenogenital syndrome
      c. adrenal adenoma or carcinoma
    2. Ovarian
      a. Stein-Leventhal or Polycystic Ovary syndrome
      b. virilizing ovarian tumors
      c. metastatic carcinoma to ovary
    3. Pituitary
      a. Cushing's syndrome
      b. acromegaly
    4. Intersex problem
      true or pseudohermaphroditism
  B. Congenital
    1. Hypertrichosis lanuginosa
    2. Cornelia de Lange syndrome
  C. Miscellaneous — starvation, anorexia nervosa, juvenile hypothyroidism, acrodynia, porphyria cutanea tarda, Achard-Thiers, Morgagni's syndrome

metabolism of adrenal steroids that favor production of androgens over glucocorticoids. In the former, the defect is complete so the patient usually presents with ambiguous genitalia at birth or with virilization in early adolescence. Adult adrenogenital syndrome is only a partial enzymatic dysfunction so that problems may not appear until adult life. As seen in *Figure 1*, there are many steps where errors in steroid biosynthesis may occur. More than 90 per cent of the causes of congenital adrenal hyperplasia may be attributed to a defect at the 21-hydroxylase step which then favors the production of androgens. These patients will show elevations in 17-ketosteroids and pregnanetriol (17-OH-progesterone metabolite) if measured in a 24-hour collection of urine. With adrenal neoplasia, dedifferentiation of tissue results in acquired enzymatic defects that may favor androgen production and result in virilization.

Excess body hair due to ovarian disorders falls into two main categories. The first, ovarian tumors — both malignant and benign — may be associated with virilization. The clinical presentation is that of a young female with a history of sudden onset of virilization, amenorrhea, and an ovarian mass usually palpable on pelvic examination. The serum androgen level, particularly testosterone, is usually quite elevated. The second ovarian category, the Stein-Leventhal syndrome, or polycystic ovary disease, is an ill-defined disease state in which the patient presents with hirsutism, irregular menstrual cycles or amenorrhea, ovulatory dysfunction, and infertility. Of note is that these patients rarely present with true virilization. It is presumed that there is an



over-production of androgens such as androstenedione from the ovary or a primary abnormality in hypothalamic-ovarian feedback that is responsible for this disorder. The ovaries may be enlarged with thickened capsules and many cysts. However, this finding on pelvic examination or laparoscopy is present in only 50 per cent of patients with the polycystic ovary syndrome. Hirsutism is found in 95 per cent of these patients, infertility in 75 per cent, and amenorrhea in 50 per cent. Considering the spectrum of the clinical findings, this may be a hard diagnosis to make without direct visualization and biopsy of the ovaries and an endocrine evaluation.

Other endocrine disorders include Cushing's disease and acromegaly but in many of these instances excess body hair may not be the presenting symptom. The intersexual problems include the true hermaphrodites and male pseudohermaphroditism where, in the latter, the gonadal sex is different from the external phenotype.

Idiopathic hirsutism is a diagnosis of exclusion once all other possibilities have been considered. The pathophysiology of this has not been well defined but several mechanisms are possible. The active androgen may not be testosterone but dihydrotestosterone with more of the latter being found in the hirsute individual. There is other evidence that there is simply end organ hypersensitivity or lowered threshold to any androgen. Elevation of the free non-protein bound fraction of testosterone and an increased turnover of testosterone have been mentioned as possible mechanisms (serum testosterone routinely measures both the free and the bound together).

Evaluation

The evaluation of the hirsute female may follow the algorithm as shown in *Figure 2*. A good history and physical examination is imperative. The family history is important as hirsutism may be very common in certain families. In point of fact, polycystic ovary disease can occur as a dominantly inherited disorder. It is therefore important to find out if there are relatives who have undergone any ovarian evaluations in the past. An abrupt change in hair growth and pattern may be an important clue to the presence of an ovarian or adrenal tumor. Abnormal menstruation or infertility will also suggest an endocrine disturbance. The physical examination should evaluate amount and type of hair as well as signs of virilization. A careful pelvic examination should be done to evaluate ovarian position and size.

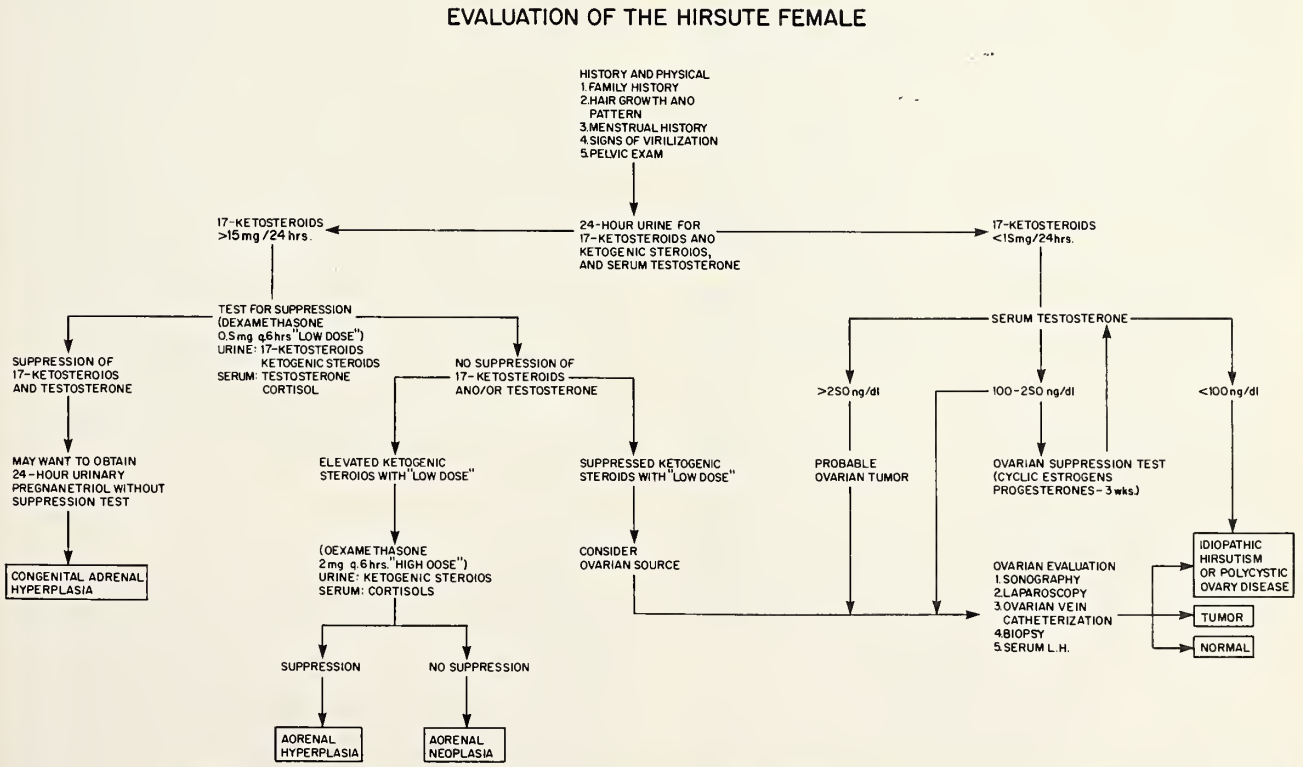


Figure 2. Diagnostic flow chart for evaluation of the hirsute female. See text for details.

Laboratory Testing

In many cases the laboratory evaluation of the hirsute female has been unrewarding because there may be only subtle abnormalities in steroid metabolism which are not reflected in the routine tests available. The 24-hour urinary collection for 17-ketosteroids reflects androgen production, usually from the adrenal gland. A probable disorder of the adrenal is indicated by an elevation above 15 or 20 mg. Ovarian disorders rarely affect the level of 17-ketosteroids to a significant degree. The serum testosterone will usually reflect ovarian hypersecretion of androgens, but it can point to the adrenal — particularly if 17-ketosteroids are also elevated. Should 17-ketosteroids be elevated, a suppression test with low dosage dexamethasone (0.5 mg po/six hrs for two days) should be performed. A 24-hour urine collection for steroids as well as serum testosterone should be obtained on the second day of this test. If suppression occurs, the diagnosis of congenital adrenal hyperplasia may be considered. If no suppression occurs, further adrenal evaluation should be done with consideration of the levels of 17-ketogenic steroids and plasma cortisols before and after high dosage (2 mg po/six hrs for two days) dexamethasone suppression. This test will help differentiate between hyperplasia and neoplasia of the adrenal gland.

Ovarian evaluation should be performed if the testosterone is elevated and adrenal steroid suppression is normal. This may include sonography, laparoscopy, biopsy, and selective ovarian-adrenal vein catheterization for androgen determinations if available. One may stop with the evaluation if the urinary steroids and the serum testosterone are normal. However, if the patient is also concerned about fertility, ovarian evaluation is mandatory as treatment of the polycystic ovary syndrome may include wedge resection of the ovary or clomiphene administration. Serum luteinizing hormone (LH) levels in these patients may be very high due to an abnormal hypothalamic-ovarian axis.

Unfortunately, there may be serious limitations to the laboratory evaluation of the hirsute female. Ovarian lesions may, in some cases, elevate 17-ketosteroids and, conversely, adrenal lesions may secrete testosterone. The absolute values of testosterone shown in *Figure 2* should be used as guidelines only, and the sequential steps used in ovarian evaluation should be individualized.

Suppressive studies with steroids or an estrogen-progesterone agent may be more confusing than helpful in terms of defining the organ and type of pathology present in some of these patients. Although it occurs rarely, elevated testosterone levels coming from an ovarian tumor have been suppressed. Therefore, if there is any question, and certainly if the testosterone level is greater than 250 mg/100 ml, laparoscopy or exploration is in order.

Treatment

Treatment of hirsutism depends on the underlying pathology. Unfortunately, most patients do not have a readily correctable endocrine cause for their hirsutism, and therapy has to be directed toward the cosmetic modalities of treatment such as electrolysis, depilatory agents, bleaching of the hair, or simple shaving. If the evaluation leads to a diagnosis of tumor of the adrenal gland or ovary, prompt surgical intervention is warranted. Following the patient with 17-ketosteroids or serum testosterone will help determine how effective surgical or medical therapy has been. Incomplete congenital adrenal hyperplasia will respond to small dosage, exogenous corticosteroid administration (usually in the form of prednisone, 5-10 mg/day); this will suppress pituitary ACTH and adrenal androgen over-production. While the patient with infertility and polycystic ovaries may require surgery, many with only hirsutism will respond in time to cyclic hormonal therapy. As should be apparent, it is often difficult to differentiate between females with idiopathic hirsutism, mild congenital adrenal hyperplasia, or polycystic ovary disease as the laboratory and clinical findings may be the same and the response to therapy may be similar. Corticosteroids should be administered with some caution in these patients as the risk-benefit ratio of such medication may not warrant their use. In patients who do not respond to low dosage corticosteroids, a course of conjugated estrogen therapy — given three weeks out of four with progesterone added the last five days of each cycle — may be helpful. With prolonged suppressive therapy, hirsutism may diminish over a period of several months to several years. Laboratory evidence of this may be reflected by decreased serum testosterone levels.

Summary

The majority of patients who present with hirsutism will be physiologically normal and will probably fall into the categories of familial causes or idiopathic hirsutism. Virilization must be ruled out by careful historical and physical examination. Initial laboratory screening should consist minimally of a 24-hour urine test for 17-ketosteroids and serum

testosterone test. Further evaluation may be done if the laboratory and clinical situation indicates its usefulness. Treatment should be directed toward the underlying cause of the problem.

Self-Assessment Questions

(More than one answer may be correct)

1. Polycystic ovary syndrome may be characterized by:
   a. Hirsutism
   b. Irregular menstrual cycles
   c. Infertility
   d. True virilization
2. The most important aspect in the evaluation of the hirsute female is the:
   a. History and physical exam
   b. Laboratory evaluation
   c. Sonogram of the ovaries
   d. Laparoscopy
3. Appropriate initial laboratory screening for the evaluation of the hirsute female includes:
   a. Serum testosterone
   b. 24 hour urine for 17-ketosteroids and ketogenic steroids
   c. Ovarian suppression test
   d. 24 hour urine for pregnanetriol
4. All of the below are signs of virilization. Which one is most diagnostic?
   a. Clitoral hypertrophy
   b. Hirsutism
   c. Deepening of the voice
   d. Amenorrhea
5. Laboratory evaluation of the hirsute female should be done routinely because:
   a. it will define the exact cause of the hirsutism
   b. it will be rewarding in most cases of congenital adrenal hyperplasia
   c. although plasma and/or urinary corticosteroids may be normal, plasma testosterone may be elevated
   d. it will definitely diagnose a primary ovarian problem.

(Answers on page 679)

Suggested Readings

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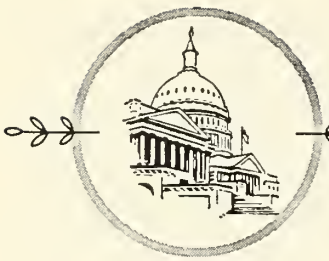
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Socio- ECONOMICS

Estate Tax Considerations When Incorporating

IT IS IMPORTANT that the professional consider the many estate tax advantages that can be realized by incorporating a practice in addition to the many income tax advantages discussed in previous articles.

Qualified Plan Life Insurance

While there are other methods available to remove personally owned life insurance from an estate, the most effective way for the professional who has a need to own substantial amounts of insurance is to have it purchased by a pension trust. Not only does this allow for deduction of premiums as a business expense but also removes the death benefit from any income or estate tax liability. It is important to note that this insurance must be payable to a named beneficiary — *i.e.* spouse or children rather than to the professional's estate — to enjoy the estate tax exclusion.

Death-Only Deferred Compensation

Many professionals affiliated with a multi-physician corporation may want to consider the installation of a death-only deferred compensation agreement. Under this arrangement an agreement can be executed with the corporation to continue paying a percentage of salary for a specified number of years to the surviving spouse. The total benefits payable under this agreement should not exceed two times annual salary from the corporation. The beneficiary of these payments will incur an income tax liability but may exclude this benefit from the estate. This arrangement may also provide significant income tax advantages to the corporation.

Stock Redemption Agreements

One of the most difficult problems facing the executors of the professional's estate is the assigning of value to corporate stock. The Internal Revenue Service's estimate of the value of stock may vary from its actual worth by tens of thousands of dollars. This can result in substantial additional estate taxes for survivors. The most effective method of avoiding this problem is to execute a mandatory stock redemption agreement with the corporation and other stockholders. Provided this agreement is an arm's-length transaction (not involving a member of the immediate family) and is binding on all parties, the IRS will accept this valuation.

Excess Group Life Insurance

Most professional corporations are already taking advantage of the tax savings that can accrue by purchasing up to $50,000 of group life insurance. However, many are not aware that significantly larger amounts can be super-imposed above the normal group insurance limits. While this additional insurance does not enjoy the income tax advantages available with normal group insurance, if properly designed these benefits may be excluded from the estate.

Further information may be obtained from Midwest Pension Planners, Mr. John E. Lyons, Plan Consultant, 1010 Merchants National Bank Bldg., Topeka, KS 66612.

Management Consultants

Rx for an Ailing Practice

Ed Note: This is the ninth in the series of articles prepared by the AMA Department of Practice Management, Division of Medical Practice. For other articles on this topic, see the following issues: November and December 1977, and January, February, March, July, September, and November 1978.

The subject of practice management has become more critical in recent years. To assist the physician in a smoother operation of his office, the Department has produced workshops for medical assistants dealing with such aspects as collections, public relations-telephone techniques and office management, as well as practice management workshops for the new physicians. The Kansas Medical Society Executive Office will present such workshops upon demand by members.

THINKING OF INCORPORATING your practice? Personnel problems getting you down? Setting up a pension or profit-sharing plan? Getting seemingly contradictory advice from your attorney, accountant, broker, and insurance man? Well, maybe it's time for you to find a medical management consultant.

Professional medical management consultants come from a variety of backgrounds — law, accounting, and business. Most consultants are organized into firms or individual consultants and, like physicians, they often specialize. But the key thing to remember is that management advisors do specialize in serving physicians and dentists, and are well acquainted with the business side of medical practices.

What can you expect in the way of advice and assistance? Firms vary, but most offer services in three broad areas: practice management, personal financial management, and accounting and taxes.

Having a consultant does not mean that you won't need the services of an attorney, accountant, or architect. But if you include your consultant in discussions with your other advisors, you will have the benefit of a valuable second opinion.

Think of it this way: if your business affairs need "doctoring," a medical consultant is like the family practitioner who can take care of most ailments. And like a family doctor, they will refer you to a specialist if you need one. Specifically, you can expect assistance in office design, accounts receivable management, tax, personnel hiring, training and policies, big equipment purchases, and the like.

What Do They Charge

Fees vary with the time and effort to do what you ask. Few bill on a daily rate basis. Most charge between $50-$100/hour, including travel time to your office and the time it takes to write reports. Rates for a complete practice survey range from $400 to $1,600, depending on type of practice. Most consultants will quote a fee before accepting a client, but if they do not, be sure to ask. You should also know that most consulting firms do not accept fees or commissions from suppliers, pharmaceutical companies, insurance companies, or other commercial interests.

Qualifications

After several years of experience, management consultants can voluntarily join a professional society and this is one way to check their credentials. These societies establish ethical standards for members and provide continuing education courses, which is as important for them as it is for the physicians.

If the consultants have "C.P.B.C." after their names, this means they have passed a comprehensive written examination on various aspects of practice and financial management. Nearly 100 medical management consultants in the United States (of approximately 500 in business) have passed the test, which is administered by the Institute for Certified Professional Consultants.

The Hirsute Female

(Continued from page 677)

Answers

1. a, b, c.
2. a.
3. a, b.
4. a.
5. b, c.

The President's Message



The holiday season is paradoxical at best.

It is a time of hustle and bustle; of addressing Christmas cards; buying, wrapping, and mailing gifts; stocking the larder; trimming the tree; cleaning the house; partying with family and friends.

It is a time of slowed professional demands — no one wants to be hospitalized over the holidays — allowing a bit more leisure.

It is a time of joy, togetherness and reflection on blessings of freedom, health, family, friends, and material assets.

It is also a time of serious problems — inflation, energy, taxes, family disintegration, drugs, and unstable moral and social values.

It is a time of loneliness, severe depression, and suicide; a time of despair for the many excluded by poverty from the traditional celebrations.

Why this polarity? Is there rhyme or reason to the holiday season or is it just a ripoff with the jingle of the cash register becoming our Christmas carol? Amidst the revelry and despair, can we find no time for reflection on the real meaning?

A long time ago, a fellow physician named Luke penned a definitive message of relevance for our age. I hope you will find some quiet time to read it during the holiday confusion, for it is a special Christmas gift for all generations.

At this special time of year, our family wishes God's blessings for you and yours, and also to your "family" at the Kansas Medical Society:

Lee Barber	Gary Caruthers
Eleanor Bell	Ramona Perez
Val Braun	Jerry Slaughter

June Stratton

They work hard in our behalf. I know you join me in extending to them and their loved ones a merry and blessed Christmas and God's blessings throughout the coming year.

Fraternally yours,

Warren E. Mayer, M.D.

President



Editorial COMMENT

Society Doctor

Fortunately for editorial writers, there is no lack of controversial material in the current medical scene. In the same sense that the medical profession is trying to put itself out of business by eliminating disease, said writers can be viewed as promoting their own removal by offering analyses and solutions to the numerous problems which, if adopted, would eliminate any excuse for their editorial presence. One of our gentle obsessions in this connection — pondered at such diverse times as while waiting on a stop light or debating whether the early morning renal alarm can be deferred until it's time to get up anyway — has been the effort to find a theme, a pattern, or at least a common denominator to account for the number and variety of such problems in the hope that it might point a way out of the contentious wilderness. So far, the one true answer has eluded us so we're not yet forced to turn to honest work but the recent chance encounter with two unrelated comments has focused us on what is at least a mechanism for viewing these problems.

The first of these comments was attributed to Dr. L. J. Henderson who taught medical history at Harvard in the first half of the century and along the way provided medical anthologists with a commendable supply of material. He observed that it was about 1910 or 1912 that the state of medical service, for the first time in history, reached the point where any given patient consulting any given physician had a better than even chance of benefiting from the experience. One might question the precise date but there is no doubt that the turn of the century brought a coalescence of medical events which, based on the wisdom and experience of the past and supported by liberal borrowings from other disciplines, has produced a fantastic progression of medical accomplishments since that time.

The second of these comments emphasizes certain changes brought about by this explosive medical experience — a sort of social sonar effect as the medical activity has bounced off the social wall and back onto the medical profession. The message came to us through an excellent publication of the School of Public Health of the University of Michigan, *Medical Care Review,* a monthly compilation of medical articles, abstracts, and reviews drawn from both lay and professional periodicals. This particular item originated as a George Will column in the Washington *Post* which referred to the thoughts of Dr. Leon Kass of the University of Chicago. While the immediate subject was the propriety of public payment for abortions, the main theme of Dr. Kass's remarks was the degree to which medical service has ranged beyond the traditional activity of overcoming an incumbent disease or trauma and returning the victim to a state of health. From that comfortably defined functional limit, medicine has come to include a compendium of services which seem to fit better in the area of social pathology than medical. The point defined by Dr. Henderson was in reality a landmark in the changing medical scene and the social pressures which have followed are a palpable sequel of this phenomenon of medical progress. It is this intrusion of social considerations into the medical service, we suggest, that accounts for the train of perplexing decisions which have confronted the physician. The population growth from its several sources, the increase in the number of aged and chronically incapacitated, the survival of the retarded of form or mind — all nonproductive or marginally so and of high maintenance cost — represent medical progress but require social solutions.

The medical profession's position recalls the proverbial Chinese philosophy that the person who saves the life of another is henceforth responsible for that person. The profession has found itself inextricably involved in dilemmas it was not prepared for but has been reluctant to share with others for fear of losing its autonomy. Perhaps this says something about one of the puzzling inconsistencies of the day,

the fact that even as increasingly strident criticism is visited on the profession's collective attitude and services, various polls — and experience — continue to indicate that the physician rates high in the public esteem and an overabundance of students continues to seek admission. Physicians, it would seem, being unprepared for the social responsibility and frequently wanting none of it, have performed ineptly in the social role while the personal image continues to hold respect.

There's a quantitative factor, too — the complaint of inadequate physician availability and improper distribution. In fact, these are largely the product of increased physician capability leading to increased physician demand leading to deficiency of physician service — whether real or relative to unrealistic expectations. It must be remembered that much of what we call increased physician capability refers to technical and auxiliary augmentation, not the ability of an individual physician to cover more miles or perform more hours.

The profession hasn't been totally ignoring of social responsibilities. The earliest manifestation of social involvement took the form of the development of public health services — whose successes, in fact, have contributed notably to the social repercussions. But the result was that the practicing group was relieved to some extent of the necessity of confronting the social problems, a condition with which it was content as long as it saw no territorial threat.

Today, there is irony in the fact that as medical successes have increased the safety of life, social trends have insisted on the assumption of risks in the name of relevance. A prime example is the current trend in obstetrics. The success of the obstetrical experience in increased safety and survival for both principals has involved several factors including hospital delivery where optimum facilities could be provided and risks were minimized. With the establishment of this safety, the social mind says we must make the process socially significant, even to moving the birthing back to the home. Increased survival and reduction of physical danger have increased society's preoccupation with the psychological and emotional elements of human discomfort.

It is not just coincidence, therefore, that this period since the turn of the century has seen the rise of psychiatry and its attendant social innovations. Initially psychiatry's growth was resisted for a number of reasons, not the least of which was that such problems do not fit easily into the conventional medical thought patterns. The organically oriented physician didn't know what to do with such cases other than to put them away some place. Because psychiatry provides a particular connection between this conventional medical function and the social utilization, its practitioners have been in the forefront of efforts to increase the social awareness of the profession, a fact that hasn't had a totally positive effect since the psychiatrist has assumed an air of superiority in such matters (supported by a whole new vocabulary) and has become much more of a socially visible animal than his organic colleague. But we hasten to add, lest we be accused of heresy right here in Mecca, that there has been serious consideration that that specialty should detach itself from the main body of medicine (cutting the cord, they would call it) and become a separate entity because of these fundamental differences. (On the other hand, as we move farther into psychopharmacology, molecular genetics and microsurgery, who knows? We may find them in the center of the fold.)

But we mustn't overlook the fiscal aspects of this situation. It would be interesting if some of our statistical experts could break down this monstrous bill being attributed to the medical profession into its component elements — the cost of actual medical service directed toward demonstrable disease as compared with that of the socially dependent indications. The abortion of a pregnancy uncomplicated except for its social implications is a prime example, as are the expenses related to the malpractice experience and its handmaiden, defensive medicine, the cost of maintaining a life after all concerned want to see it released, the expenses deriving not so much from the social impact of the medical accomplishment but produced *by* society for the medical community to take care of — to touch on a few.

Solution? This *is* the solution. Medical activity can't be separated from its social consequences and the consequences will be conditioned by the social climate of the day — meaning they will return to modify the medical conduct. In the immediate struggle to conserve what seems desirable, the medical organization is generally condemned as ultraconservative and self-seeking, but in the evolutionary range, no segment has been more altruistic and radical in its effect. If the striving for an equilibrium produces contention and friction, that, believe it or not, is progress. When the lion and the lamb lie down, it will be peaceable but they won't get much done. — *D.E.G.*

Obituaries

THOMAS C. HURST, M.D.

Dr. Thomas C. Hurst, 64, Wichita, died July 22, 1978, in Cologne, Germany.

Dr. Hurst was graduated from the University of Kansas School of Medicine in 1939 and served with the Army Medical Corps during World War II. He had practiced in Wichita since 1946.

Survivors include his wife and one son.

ROSCOE FALLS MORTON, M.D.

Dr. Roscoe F. Morton, 65, died October 30, 1978, in Arkansas City.

Dr. Morton was graduated from the University of Arkansas School of Medicine and had practiced in Arkansas City since 1949.

Survivors include his wife and two sons. A memorial has been established with UKSM-Wichita.

HENRY ST. CLAIR O'DONNELL, M.D.

Dr. Henry St. Clair O'Donnell, 85, died September 6, 1978, in Ellsworth.

Dr. O'Donnell was graduated from the University of Kansas and Washington University School of Medicine, St. Louis, and served in the Army during World War I. He had practiced in Ellsworth for 60 years, and was a former president of the Kansas Medical Society. He was active in many professional and civic organizations.

Survivors include his wife, a son, and two daughters. Memorial contributions may be made to Ellsworth County Memorial Hospital.

A. K. RATZLAFF, M.D.

Dr. A. K. Ratzlaff, 71, Goessel, died February 7, 1978.

Dr. Ratzlaff was graduated from the University of Kansas School of Medicine and had practiced in Goessal since 1939.

Survivors include his wife, three sons, and two daughters. Memorial contributions may be made to Bethel College, Newton.

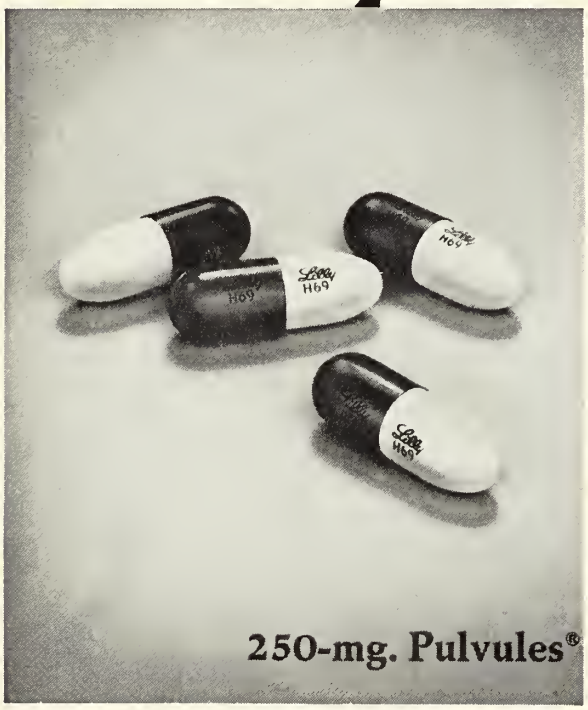
WAYNE O. WALLACE, SR., M.D.

Dr. Wayne O. Wallace, Sr., 65, Atchison, died September 27, 1978.

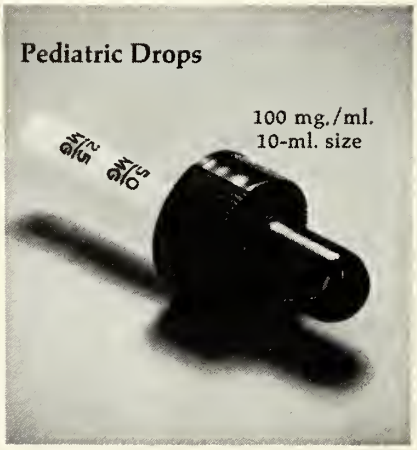
Dr. Wallace was graduated from the University of Kansas School of Medicine in 1937 and was active in many civic and professional organizations. He had practiced in the Atchison community for 40 years.

Survivors include his wife and four sons. Memorial contributions may be made to the Atchison County Heart Association, the Atchison County Retarded Citizens Association, or the Atchison First Presbyterian Church.

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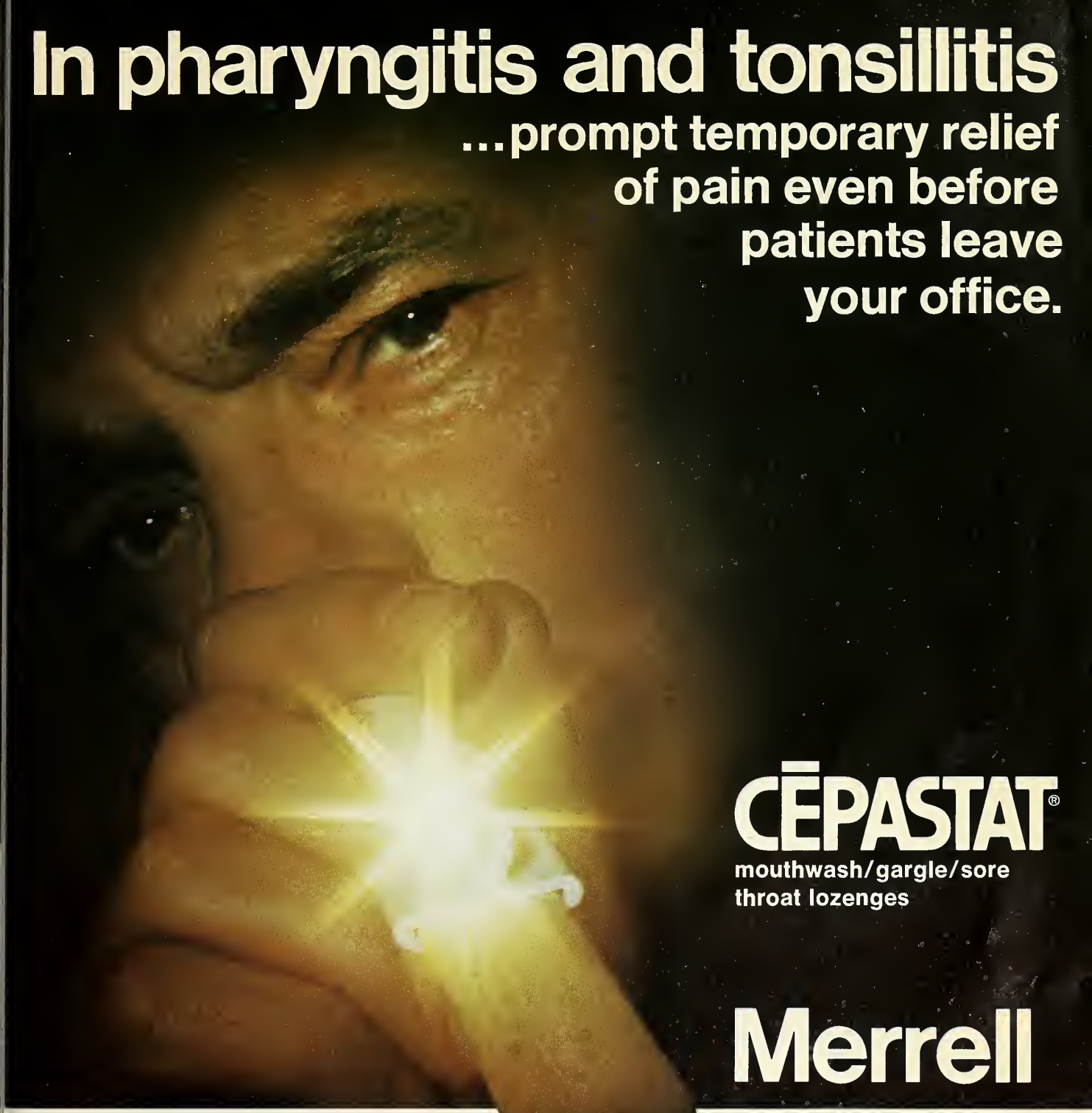
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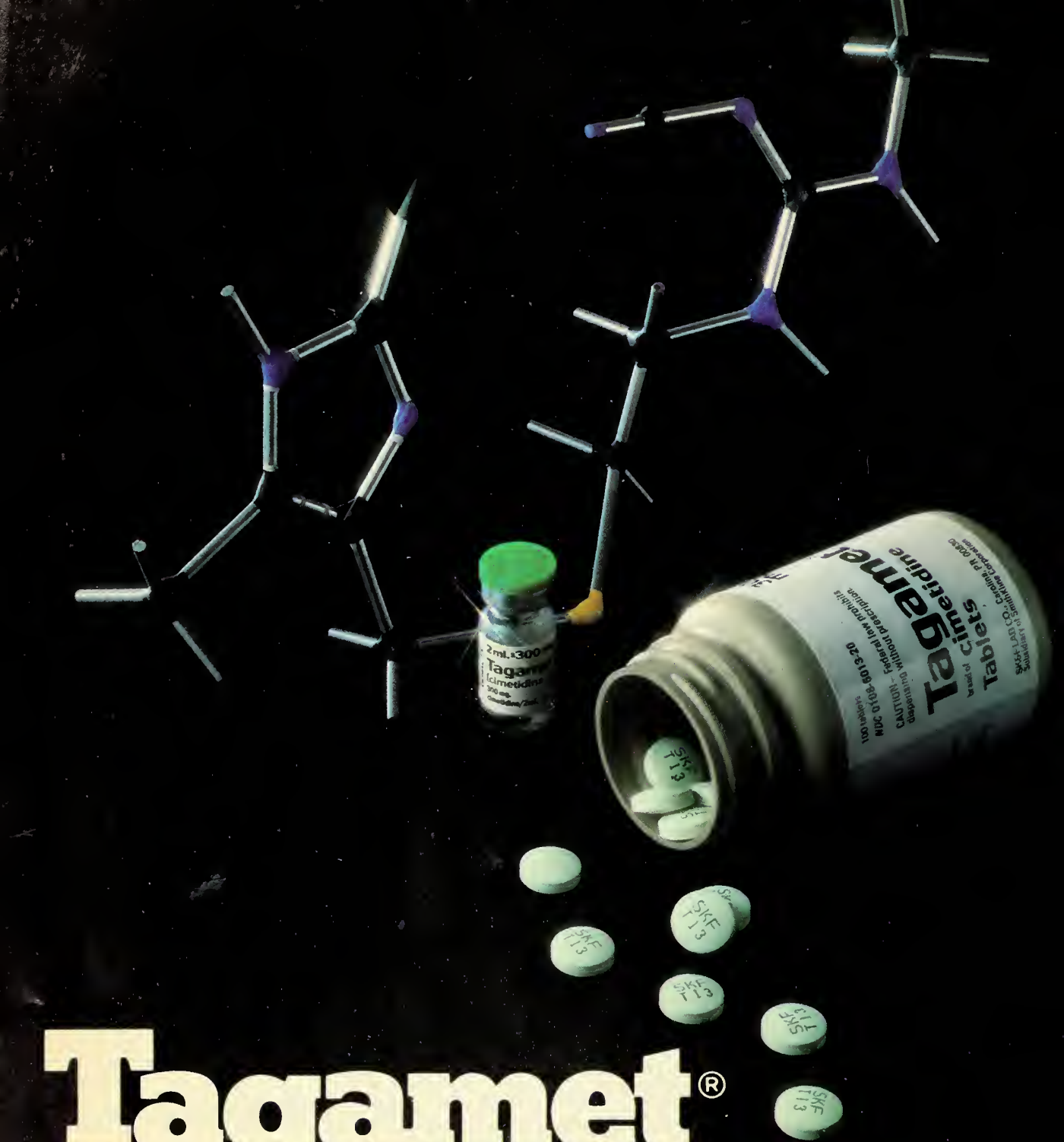
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Committee REPORTS

Cost Containment: MSSC Committee Report and Recommendations

The medical profession is deeply concerned about the rising health care costs. Throughout the country, physicians are analyzing health expenditures and seeking possible cost containment solutions. A special committee of the Medical Society of Sedgwick County was appointed to study the factors responsible for rising health care costs and to submit recommendations for cost containment solutions. Specifically, the role of the practicing physician was to be studied.

The Cost Containment Committee was appointed at the direction of MSSC Board of Directors and has met monthly since April. It has studied some of the voluminous literature on this subject and, probably more productively, it has had frank and sometimes spirited discussions on the opinions and personal experiences of the Committee members.

The most important point that the Committee wishes to make is when dealing with this kind of problem, the natural inclination and simplistic approach is to look for a villain. The first conclusions of the Committee were that medical care and its costs constitute a drama in which there are no villains but a multitude of accomplices.

The cast of the scenario includes consumer-patients; physicians, hospitals, auxiliary personnel and providers; insurance company-governmental third party-payers; educational institutions; unions and management; private organizations and agencies; and last but not least, government on every level. Each group has to deal with all the others in a myriad of complex ways on the health care stage. Each group, for its own reasons and in its own way, has been an accomplice in causing health care expenditures to increase.

Throughout these complex, and many times impersonal relationships, there is very little, if any, coordination or incentives to contain costs. Each party is primarily interested in playing its respective role in providing the highest quality and quantity of health and medical care to the public it serves.

It is essential that "rising health care costs" be precisely defined. Is it that costs are rising unjustifiably and that patients are getting "ripped off"? Or is it that the aggregate demand for and use of medical services with total costs are uncomfortably accelerating?

Certainly there have been isolated instances of fraud, and the medical profession fully supports efforts to eliminate such abuse. But this constitutes a tiny fraction of health costs.

Fees for many individual services have risen over the years, but so has the quality of services, as well as the costs of providing these services. For example, much has been made over high hospital room rates. But the hospital of today is almost as different from the hospital of 10 years ago as one of the new luxurious recreational vehicles is different from a pup tent.

Another example is the increasing billions of dollars spent for laboratory tests in this country annually; yet, through automation and better laboratory management, fees for the most common tests have risen little. In many cases, lab fees have actually declined over the years, while the quality of the tests improve.

These findings have led the committee to conclude that, for the most part, the patient is getting a fair deal for his money and that the individual unit costs of medical services are not unreasonable.

Long-term cost containment without sacrificing quality care can only come by curtailing exuberant proliferation and overuse of health and medical services, and not by singling out and attacking individual unit service costs.

Why is the use of health services increasing so rapidly? This is where everyone in health care is an accomplice. The physician has been educated to place the care of the patient above all else. The doctor not only wants patients to have the best, which may become synonymous with most, but he is afraid to offer less for fear of losing the esteem of patients and peers or, even worse, of encountering a devastating malpractice suit. There are few incentives in the present system for the doctor to do otherwise.

The patient has been led to believe that first class health care on demand and in quantity is his right, regardless of ability to pay or the nature of his complaint. Since the vast majority of citizens are now covered by some type of insurance, the cost is somebody else's problem.

Hospitals are in a fiercely competitive marketplace. The competition is obviously not cost, but quality and quantity of services. Hospitals don't have patients. Doctors have patients. To keep their doors open today, hospitals have to satisfy both. The way to do this is to offer the latest, the best and the most. This seems what most people want, and somebody else pays the bill.

Insurance companies rightfully like to sell policies, but the promotional trend has been to ever greater first dollar coverage and fewer restrictions that decrease patient and provider responsibility. When the resultant overuse causes decreased profits, increased premium rates seem to be the answer.

Educational institutions glorify science and technology. Too often they neglect to teach students (tomorrow's doctors) the distinction between what may be appropriate in a limited research academic setting and what is reasonable quality health care in the community.

And then there is government. The committee thinks the government on all levels has pursued policies leading to general inflation that is unacceptable to most citizens. These inflationary pressures are intensified in health care, which is probably the number one highly educated labor and technology intensive industry.

More specifically, government creates new health programs that inflame the expectations of people without reasonable projections as to what these programs will cost or if the benefits justify the expenditures. It becomes a bloated bureaucracy whose activities often generate an expansion of its own empire and create a nightmare of rules, regulations and paperwork that are often duplicative, confusing, and inflationary. It is ironic that government is usually careful to exclude its own health care facilities from these regulations.

Such government actions drive up overhead costs which, in turn, increase the cost of providing medical services, lead to decreased productivity, and force many providers to discontinue participation in the government-sponsored programs. This, in turn, forces patients to obtain care from inappropriate sources.

What, then, are some of the possible solutions?

I. PHYSICIANS:

Physicians play an important role in shaping health care costs. While receiving only approximately 18-20 per cent of the total health care dollar for professional services, they direct where some 80 per cent will be spent through hospital admissions, diagnostic tests, nursing homes, prescriptions, etc. Practicing physicians, residents, and medical students need more exposure to medical care economics.

A. RECOMMENDATIONS:

1. Teaching programs need to stress medical care economics, especially as it relates to laboratory and diagnostic testing.
   a. Laboratory tests and other diagnostic procedures should be used to confirm the diagnosis already established through the patient's history and physical examination, rather than for the purpose of obtaining information on which a diagnosis is to be established.
   b. Residents, medical students, and practicing physicians should question more often as to why a specific laboratory test or diagnostic procedure was requested.
2. A list of costs for various laboratory tests and diagnostic procedures should be posted in clinic areas, resident conference rooms, and other specified areas of each hospital.
3. Lectures on cost for various laboratory tests, diagnostic and special procedure, and cost of hospitalization per day in different sections of the hospital should be given at noon conferences at each hospital at least every month.
4. Direct feadback of hospital costs should be provided to residents and practicing physicians by sending them randomly a copy of their patient's hospital bill in

itemized form. This would also provide discussion material at noon conferences which would be reserved in part for medical cost containment discussions. This would acquaint physicians with charges for hospital services continually and would allow for a retrospective review of the management of the case from a qualitative and quantitative point of view.

5. Physicians should review their individual office practices regarding fees and use patterns to decrease and/or contain medical care costs.
6. Excerpts or summaries of articles regarding medical economic topics should be carried regularly in the MSSC's monthly newsletter.
7. Timely subjects regarding medical care economics should be included as part of the MSSC's monthly membership meetings.
8. In cooperation with local hospitals, physicians must develop more effective review programs. Physicians should review each other's practice patterns for quality, necessity, and appropriateness of where services were provided.

II. PATIENTS:

Citizens need to be educated and motivated concerning meaningful preventive medicine. This means proper diet, exercise, and refraining from all types of overindulgence. There are many patients in hospitals and doctors' offices who need not be there were they to lead a more healthful life-style.

Patients need to realize that whenever they request unnecessary hospitalizations, pressure the physician for unnecessary tests and treatments, or seek attention for trivial complaints, they ultimately will pay.

A. RECOMMENDATIONS:

1. Include as a part of the required school curriculum a unit of health education developed by local boards of education in consultation with local medical societies for all grade levels, kindergarten through 12th grade.
2. All persons and families should establish a patient-physician relationship with a primary care physician. This physician will be able to care for approximately 90 per cent of medical needs. These relationships should help reduce the inappropriate use of hospital emergency rooms.
3. Citizens must understand that their abuse or inappropriate use of public and private medical programs increase the overall cost of medical care.
4. Patients should be made to feel free to discuss openly with their physician, insurance agent, and hospital administration questions on costs and charges.
5. The MSSC should develop informational leaflets informing patients about health measures that could lead to improved health and reduce health care spending. These could be used as monthly statement stuffers.
6. Citizens should be informed that some types of so-called preventive medicine programs that are not directed towards specific health problems or health maintenance are not always cost effective. In fact, they often increase health care costs.

III. HOSPITALS:

Hospitals will have to learn that everyone can't be everything to everybody all the time. They must say "no" when necessary. They should develop their own areas of services that are needed in the community, while cooperating more and sharing with each other to avoid unnecessary duplication.

A. RECOMMENDATIONS:

1. The members of the medical profession, individually and collectively, should support and cooperate with the current Hospital Voluntary Cost Control Program being instituted by the AHA, FAH, and AMA.
2. Hospital administrations should examine cost impact of all requests and recommendations for new services. Decisions should be based on the community's medical needs and improvement of overall quality of care, rather than to satisfy interests of special groups.
3. Existing hospital utilization review and medical audit committees should be reviewed for their goals and effectiveness. These committees should look more closely at need for service and economics of care delivered, as well as assessing quality of care.
4. Determine, in cooperation with a com-

mittee to be appointed by the MSSC, if there is inappropriate use of hospital beds in the Wichita community.

5. Cooperate with recommendations concerning physicians under I-A 2, I-A 3, I-A 4, and I-A 6 of this report.
6. Review, update, and coordinate with all other local hospitals' existing policies that would allow data from out-patient workups to be included on the patient's hospital medical records when performed within a reasonable time prior to admission.

IV. INSURANCE COMPANIES — THIRD PARTY PAYERS:

Currently, there are few, if any, incentives for insurance companies to be seriously concerned with controlling health care cost. When expenditures increase, they are recovered by passing them on to the public through premium increases.

A. RECOMMENDATIONS:

1. The committee strongly recommends reversal of the trend to first dollar coverage which absolves everybody of direct, although certainly not of indirect, financial responsibilities. A system of reasonable deductibles with co-insurance for all medical care but with expanded catastrophic coverage, we feel, would dramatically decrease unnecessary use.
2. The insurance companies should encourage determination of premium rates on a basis that would take into consideration the individual or group's life-style. They should consider, also, establishment of a built-in economic incentive for individuals and groups who exhibit appropriate or lowered utilization.
3. The insurors should provide medical societies' insurance and/or utilization review committees with data identifying those physicians whose use patterns deviate significantly from those of other local physicians of similar training. Similar patient data should also be made available to these insurance and/or utilization review committees.
4. Insurors should provide management with data regarding the group's health care utilization. This information should be used in connection with the company's employee health education programs. These programs should include discussions on cost containment, utilization, healthful living, etc. Individual employee identity must be protected. If such educational programs are not in existence, they should be organized and implemented.
5. Third party payers should provide coverage for ambulatory care and outpatient work-ups at settings based on medical necessity. This should lower in-hospital use and, conversely, increase office and outpatient use. This should also reduce patient pressure for unnecessary hospitalization.
6. They should require patients who obtain medical care at hospital emergency rooms which is not of an emergency nature to pay a higher co-insurance or out-of-pocket amount; for example: $10-$15 per visit.
7. Insurance policies should be written in language understandable to the average person. Insurance company representatives should work closely with employee group leaders and management regarding proper use of services, etc.
8. All insurance companies writing health and medical coverage should adopt the concept of "coordination of benefits." This should eliminate duplication of payment from overlapping coverage whereby patients profit by seeking medical care.

V. GOVERNMENT:

Government simply needs to become more competent and responsible. Government activities have not developed solutions but rather have been an integral part of the problem. The existing laws, rules, and regulations should be reviewed at all levels of government to determine if they are accomplishing their intended purpose and if they are cost effective. If not, they should be modified or eliminated. It appears that the government's solution to problems is writing more rules and regulations. Many are confusing, duplicative, contradictory, and, in the end, create more problems than they solve.

For fiscal year 1979, it has been estimated by Murray L. Weidenbaum's Viewpoint on Estimating Regulatory Costs, Journal on Government and Society, May/June, 1978, that the "administrative costs of federal regulation (paid by the taxpayer) and the compliance costs (gen-

erally passed on to the consumer in the form of higher prices) may top $100 billion."

A. RECOMMENDATIONS:

1. Medical services available through government sponsored programs should allow the patient free choice of hospitals and providers, with reimbursement to be on the basis of reasonable charges. At the present time, reimbursement for provider services through government sponsorship promotes the use of hospital services such as emergency rooms and encourages what has been called "mill-type operations." Care provided in these settings is more costly than when provided in the physician's office. This type of reimbursement also forces many physicians not to participate in these programs.
2. Kansas Statute 77-416, adopted by the Legislature in 1977, should be amended so a specific party would be assigned the responsibility of reviewing the reasonableness and cost effectiveness of all proposed rules, regulations, and amendments. A penalty should be provided if the intent of this statute is not complied with.
3. Lawmakers should not pass broad, permissive, enabling legislation that delegates to government employees the responsibility of writing rules and regulations that often distorts the intent of the original legislation.
4. Although the intent of most laws, rules and regulations may be well meaning, the required paper work, duplication of program administration, etc., often defeat successful implementation. Many programs involving administration at the federal, state, and local levels bring about costly duplication of agencies and empire building. Compliance with the program's regulations increases overhead costs, creates ill-will and unsatisfied consumers, and encourages withdrawal of providers. Efforts should be made to streamline the administration of all government programs. General guidelines should be developed at the federal level, with program administration resting with state and local governments. Duplicating and unnecessary regulations should be eliminated.
5. Existing government and voluntary accrediting and licensing bodies should review existing programs to eliminate rules and regulations which are not cost effective or do not relate to improvement of quality medical care. All such programs should be coordinated to eliminate duplication and fragmentation of activities. All future rules and regulations should be kept to a minimum and scrutinized closely for cost effectiveness.
6. Alternative health care delivery mechanisms should continue to be explored; however, the pluralistic system should be preserved. Government financial subsidization should not favor one over the other. Each should succeed or fail based on its own merits.
7. The Federal Trade Commission should initiate steps that would lead to rescinding court decisions that have eliminated local medical societies' fee review committees. These committees provided patients, insurance companies, etc., with a mechanism to review questions about professional charges.

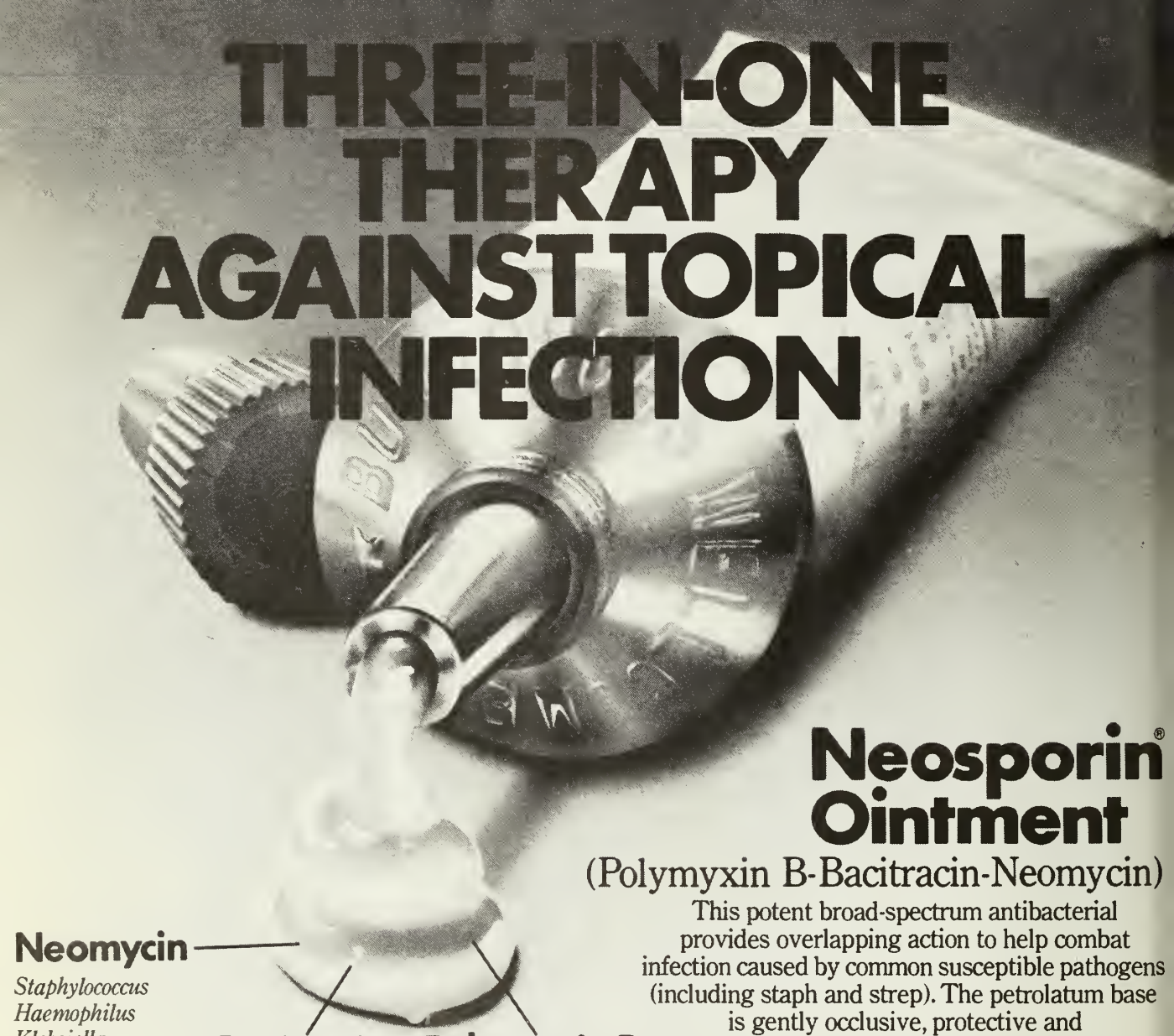
Conclusion

Members of the MSSC Cost Containment Committee conclude that rising health and medical care costs are a result of many complex factors involving a multitude of individuals and organizations. If cost containment efforts are to be successful, solutions cannot be directed "piece meal" at specific segments of the health care delivery system but must address simultaneously, and in a cooperative manner, all involved components.

The committee respectfully submits this report and recommendations, believing it has performed to the best of its ability the charges outlined by the Society's Board of Directors. The committee urges the Board to move forth with meaningful implementation.

Committee Members: William Bruner, M.D., Chairman; David Brown, M.D.; Leo Cawley, M.D.; Ronald Davis, M.D.; Krishan Goyle, M.D.; James Loeffler, M.D.; William C. Loewen, M.D.; Robert T. Manning, M.D.; Merri McMahon, M.D.; Philip A. Miller, M.D.; Steven V. Moore, M.D.; Michael Randles, M.D.; Ivan E. Rhodes, M.D.; Timothy Scanlan, M.D.; C. C. Schopf, M.D.; Richard Skibba, M.D.

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Personalities —IN KANSAS MEDICINE

A recent series of medical lectures at Colby Community College featured a presentation by **Asher Dahl,** Colby, on fractures, and one by **George Marshall,** Colby, on arthritis.

Gerald Nelson, Wichita, was a participant in a short term clinical mission to the Dominican Republic sponsored by Medical Group Missions program of the Christian Medical Society.

A plaque recognizing 32 years of service to the community was presented to **George W. Hammel,** El Dorado, at a recent meeting of the Butler-Greenwood County Medical Society.

Tell Copening, Iola, was the featured speaker at a recent meeting of the Allen County Chapter of American Medical Assistants.

Thomas Kneidel, UKSM-Wichita, was a featured lecturer for the Fourth Annual Harper Seminar Series and Mobile In-Service Education Program.

Daniel Hinthorn, UKSM-Kansas City, recently attended a meeting of the Royal College of Physicians in London, England.

Herman Solomon, UKSM-Wichita, presented a section on "Geriatric Skin Problems and Other Dermatology Issues" as part of the Third Annual Kinsley Seminar Series.

Phillip Olsen, El Dorado, spoke on Arthritis at a recent meeting of the Butler County Division of the Kansas Chapter, Arthritis Foundation.

Earnest McClellan, Wichita, presented the program for a recent meeting of Region I, Emergency Medical Services Development Council in Hoxie.

V. W. Steinkruger, Hugh J. Woods, and **R. G. Sheppard** were accorded special recognition at a recent appreciation night for physicians in Smith Center.

Richard Siemens, Lyons, was featured speaker at the first session of a series of education classes for arthritics.

R. P. Quackenbush, St. John, recently participated in a one-week hospital training session to update knowledge of arthritic diseases.

Michael R. McGuire, Shawnee Mission, has been admitted to Fellowship in the American College of Cardiology.

Leslie Cobb, Mulvane, discussed cardiovascular emergencies at a recent meeting of Emergency Medical Services of the Mulvane Civil Defense. Dr. Cobb conducts regular recertification training for EMS.

Donald D. Goering, Salina, was named a runnerup for Family Doctor of the Year award presented annually by *Good Housekeeping* magazine and the American Academy of Family Physicians. He was cited for his role in establishing a Department of Family Practice at UKSM.

Donald Bebak, Wichita, spoke on the topic of abortion from a doctor's viewpoint at a recent meeting of the Hutchinson chapter of Right to Life.

Herbert Fransen, Newton, discussed major surgery patients at the first Doctors' Seminar of the New Learning Exchange.

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At a Death and Dying Seminar recently sponsored by the United Methodist Church in Sterling, **Tom Simpson** spoke on "A Physician Looks at Death."

C. A. Nystrom, Topeka, addressed the Men's Breakfast recently in Downs.

Robert Enberg, Hays, teaches infant-care courses throughout Kansas and has been instrumental in the establishment of the Northwestern Kansas Infant Transport System.

D. Cramer Reed, Wichita, presented the first program on community health education sponsored by McPherson Memorial Hospital Auxiliary Foundation.

Frank Masters, Kansas City, has been chosen 1978-79 President-Elect of the American Society of Plastic and Reconstructive Surgery.

Richard Beach, Topeka, addressing the Retail Divison of Downtown Topeka, warned of increased costs and problems that could be generated by serving liquor in restaurants in Kansas.

Larry Miller, Wichita, presented a program on "Family Practice Residency" at a recent meeting of the Butler-Greenwood County Medical Society.

Based on his contribution to the district office, **W. Reese Baxter,** Salina, has received the Medical Services Award from the Kansas Rehabilitation Association.

Roy Neil, Hays, presented a seminar on Drug and Alcohol Abuse as part of A Family Forum sponsored by the Hays Council of Church Women United.

S. M. Steinzeig, Kansas City, spoke on "What Is Coronary Disease?" at a recent public meeting sponsored by Mended Hearts.

John W. Travis, Topeka, has been named president-elect of the American Society of Therapeutic Radiologists.

John Cody, Hays, spoke on "Stress — We All Have It!" at the annual public meeting of the Ellsworth County Mental Health Association.

"Heredity and Diabetes" was the topic addressed by **Richard A. Guthrie,** Wichita, at a recent meeting of the Harvey County chapter of the American Diabetes Association.

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Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. Oral—Adults: Mild and moderate anxiety and tension, 5 or 10 mg t.i.d. or q.i.d.; severe states, 20 or 25 mg t.i.d. or q.i.d. Geriatric patients: 5 mg b.i.d. to q.i.d. (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl) Capsules, 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10. Libritabs® (chlordiazepoxide) Tablets, 5 mg, 10 mg and 25 mg—bottles of 100 and 500. With respect to clinical activity, capsules and tablets are indistinguishable.

synonymous with relief of anxiety

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Roche Products Inc.
Manati, Puerto Rico 00701

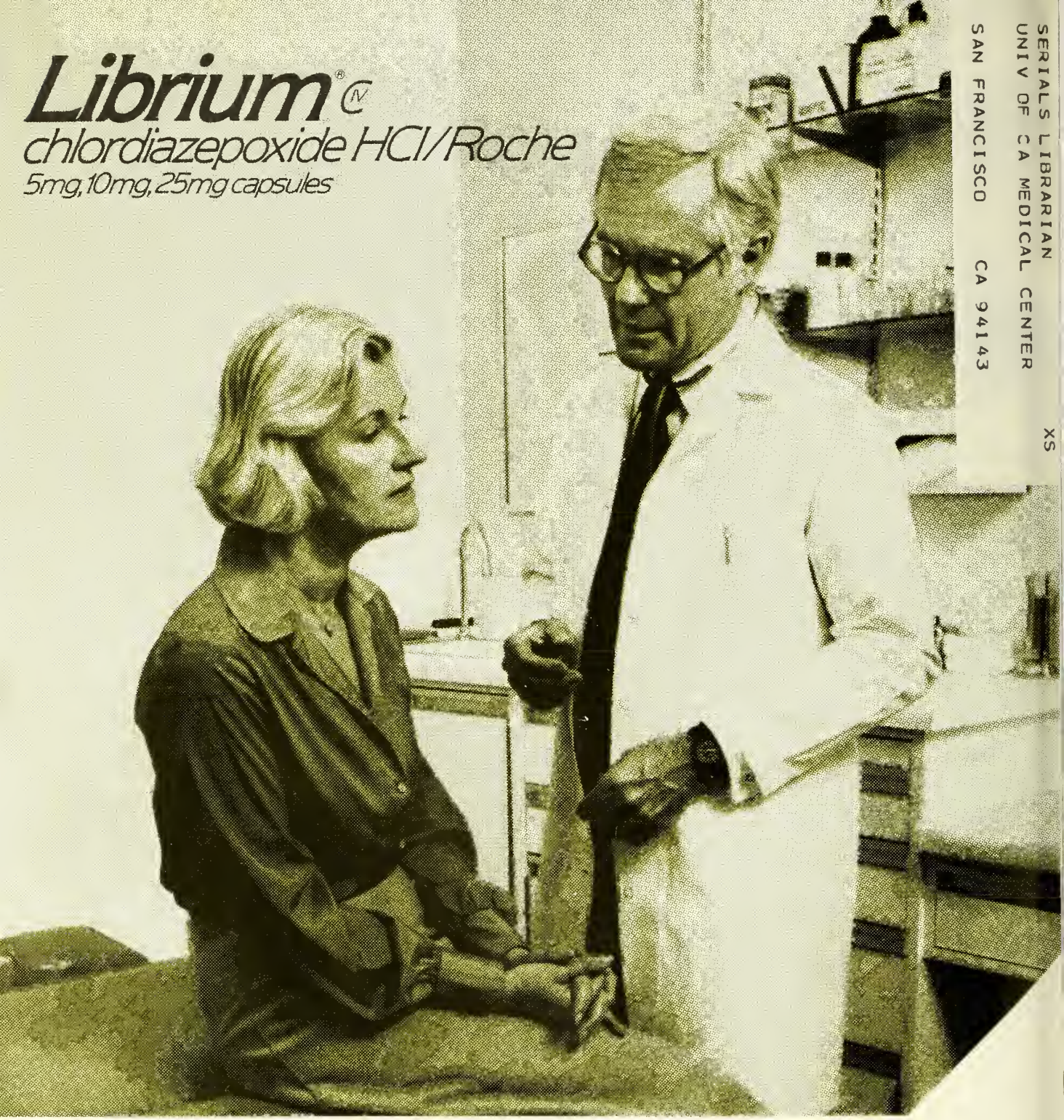
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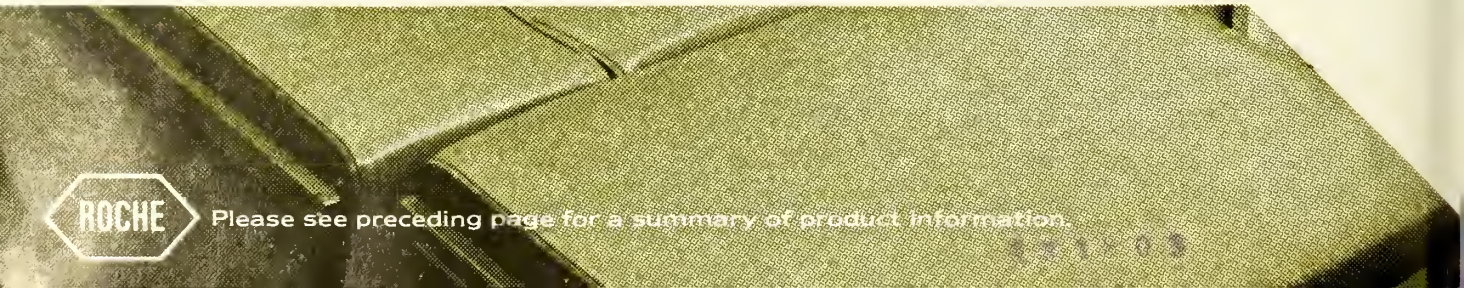
Librium® C[IV]

chlordiazepoxide HCl/Roche

5mg, 10mg, 25mg capsules



synonymous with relief of anxiety



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Please see preceding page for a summary of product information.

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